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SOME BYGONE OPERATIONS IN SURGERY.

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IX. A CASE OF STRANGULATED UMBILICAL HERNIA. QUEEN CAROLINE OF ANSPACH.

CAROLINE OF ANSPACH (1683–1737), wife of George II and Queen of England, was a slim and good-looking girl when she was married, and throughout her life was endowed with more than a modicum of common sense. The mother of seven children, she complained of getting fat as she grew older. In 1723 she developed an umbilical hernia after the birth of her sixth child, but kept the fact to herself so strictly that the King did not know of its existence for many years. It increased steadily in size, and at last became strangulated, causing her death fourteen years after its first appearance. Lord Hervey in his memoirs gives a lively account of the final scenes, and as he was a favourite in constant attendance at Court his story is probably accurate in every particular. He says :—

" On Wednesday, the 9th. November (1737) the Queen was taken ill in the morning at her new library in St. James' Park. She called her complaint the colic, her stomach and bowels giving her great pain. She came home took Daffy's Elixir by Dr. Tesier, the German, and house-physician's advice but was in such great pain and so uneasy with frequent retchings to vomit, that she went into bed. However when the clock struck two and the King proposed sending Lord Grantham, to dismiss the company and declare there would be no drawing room, she, according to the custom of the family, not caring to own or at least to have it generally known how ill she was, told the King she was much better, that she would get up and see the company as usual. As soon as she came into the drawing room she came up to Lord Hervey and said ; 'Is it not intolerable at my age to be plagued with a new distemper ? Here is that nasty colic that I had at Hampton Court come again.' The Queen twice this summer at Hampton Court had been seized with a vomiting and purging which had lasted in the most violent manner for three or four hours and then left her so easy and well that she had played the same night in the drawing-room as usual and had talked almost with the same cheerfulness. This made Lord Hervey less alarmed than he otherwise

would have been for she looked extremely ill. Lord Hervey asked her what she had taken and when she told him, he replied 'For God's sake, Madame go to your own room, what have you to do here.' She then went and talked a little to the rest of the company and coming back to Lord Hervey, said, 'I am not able to entertain people.' 'Would to God the King would have done talking and release you,' replied Lord Hervey. At last the King went away, telling the Queen as he went by that she had overlooked the Duchess of Norfolk. The Queen made her excuse for having done so to the Duchess of Norfolk, the last person she ever spoke to in public and then retired, going immediately into bed, where she grew worse every moment.

"At seven o'clock when Lord Hervey returned to St. James's he went up to the Queen's apartment and found her in bed. The Queen asked Lord Hervey what he used to take in his violent fits of the colic; and Lord Hervey, imagining the Queen's pain to proceed from a goutish humour in her stomach that should be driven from that dangerous seat into her limbs, told her nothing ever gave him immediate relief but strong things. To which the Queen replied; 'Pshaw! you think now like all the other fools that this is the pain of an old nasty stinking gout.' But her vomitings, or rather her reachings, together with such acute pain continuing in a degree that she could not lie one moment quiet, she said about an hour after 'Give me what you will, I will take it.' He fetched some snakeroot and brandy and asking Dr. Tesier who was in the outward room, whether he might venture to give it to her, Dr. Tesier, who was naturally timid and made more so by the manner in which he had been talked to in the King's illness last year, said the Queen's pulse was very high and feverish and as she was unused to drinking anything strong, he could not affirm that this very strong cordial would do her no hurt. Lord Hervey then asked him if he should propose to the King to call in another physician, and if he had any objection to Broxholme; and Dr. Tesier saying he wished it extremely but did not dare to propose it himself to the King. Lord Hervey told Princess Caroline what had passed and would, if she approved, propose to the King that Dr. Broxholme might be called in. Dr. Broxholme was immediately sent for and when he came Tesier and he agreed to give the Queen some snake-root with Sir Walter Raleigh's cordial, but this cordial being long in preparing and Ranby, house-surgeon to the King, a sensible fellow, telling Lord Hervey that insisting on these occasions upon a cordial with this name or t'other name was mere quackery and that no cordial was better than another but in proportion to its strength some usquebagh was immediately given to the Queen who kept it about half an hour but then brought it up. Soon after the snake-root and Sir Walter Raleigh's cordial arrived from the apothecary's, it was taken and thrown up about an hour after. All these strong things had, without easing the Queen's pain, so increased her fever that the doctors ordered Ranby to bleed her twelve ounces immediately. She took a glister but it came from her just as it went into her. The King inconveniently both to himself and the Queen lay on the Queen's bed all night in his nightgown where he could not sleep nor she turn about easily.

"Early in the morning of Thursday the 10th. the Queen was blooded twelve ounces more on which her fever that had been very high all night

QUEEN CAROLINE OF ANSPACH

abated and everybody but herself thought she was better. The vomiting was suspended for a few hours but nothing passed downwards and two glisters she took returned immediately and pure. This evening whilst the Princess Caroline and Lord Hervey were alone with the Queen, she complaining and they comforting, she often said 'I have an ill which nobody knows of' which they understood to mean nothing more than that she felt what she could not describe and more than anybody imagined. This night two more physicians were called in Sir Hans Sloane and Dr. Hulse who ordered blisters and aperients. These came up like every other thing soon after she had swallowed it and the blisters, though a remedy to which the King and Queen had often declared themselves very averse, were put upon her legs. At six o'clock on Friday morning the Queen was again blooded upon which her fever went almost entirely off; but the total stoppage and frequent vomitings still continued. The whole Friday the Queen grew worse almost every hour. The Princess Caroline went to bed at night in her own apartment; the Princess Emily sat up in the Queen's bedchamber; Lord Hervey lay on a couch in the next room and the King had his own bedding brought and laid upon the floor in the little room behind the Queen's dressing room and lay there until about four o'clock on Saturday morning when the Queen complaining more than ever of the racking pains she felt in her belly and of a throbbing, he was called and all the physicians immediately sent for.

"When the King came into the room to the Queen he whispered her, and, as he said afterward, told her he was afraid her illness proceeded from a thing he had promised never to speak of to her again; but that her life being in danger, he could not answer it to her, himself, or his family not to tell all he knew. She begged and entreated him with great earnestness that he would not and spoke with more warmth and peevishness than she showed at any other minute during her whole illness. However he sent for Ranby the surgeon and told him he apprehended the Queen had a rupture at her navel and bid him examine her. The Queen carried her desire to conceal this complaint so far that when Ranby came to feel her she laid his hand on the pit of her stomach and said all her pain was there; but Ranby slipping his hand lower, kept it there in spite of her some little time and then without saying one word to the Queen went and spoke softly to the King at the chimney upon which the Queen started up and sitting in her bed said to Ranby with great eagerness 'I am sure, you lying fool, you are telling the King I have a rupture.' 'I am so,' said Ranby, 'and there is no more time to be lost; Your Majesty has concealed it too long already; and I beg another surgeon may be called immediately.' The Queen made no answer but lay down again and turned her head to the other side, and as the King told me he thinks it was the only tear he saw her shed whilst she was ill. The King bid Ranby send immediately for old Busier the surgeon whom, though fourscore years old the King and Queen had a great opinion of and preferred to every other man of his profession. Busier not being immediately to be found and the King very impatient he bid go and bring the first surgeon of any note and credit he could find. When Ranby returned he brought one Shipton with him, a City surgeon and one of the most eminent and most able of the whole profession. By this time, too, Busier arrived and these three

attended her constantly. After they had examined the Queen, they all told the King she was in the utmost danger. Busier proposed the operation of cutting a hole in her navel wide enough to thrust the gut back into its place; which Ranby opposed, saying that all the guts upon such an operation would come out of the body into the bed, and that he thought he felt at the bottom of the swelling (which was of an immense size) a softness which he took to be a disposition to make matter and which they might encourage by warm fomentation till the swelling might break of itself or at least allow them by a slight touch of the lancet to open it without danger, Shipton inclining to Ranby's opinion, this method was pursued. About six o'clock this Saturday evening the surgeons lanced the swelling just at her navel and let out some matter but not enough to abate the swelling in any material degree or give them any great hope of her recovery.

"About four o'clock on Sunday morning the 13th, the Queen complaining that her wound was extremely painful and desiring to have it dressed, Ranby and Shipton were called in to her and upon opening the wound declared it had already begun to mortify. Hulse, whose turn it was that night to sit up, was sent for into the Queen's bed chamber and acquainted by the surgeons with the situation she was in. Hulse came to the Princess Caroline and told her this terrible and dreadful news upon which she bid him and Ranby go immediately and inform the King. When Hulse and Ranby came back to Princess Caroline (the King being already up and gone to the Queen) Princess Caroline and Lord Hervey asked Hulse if there was no possibility left of her recovery and he answered 'None.' Lord Hervey then asked Ranby if they were never deceived in the signs of a mortification, to which Ranby shaking his head, replied; 'We know them but too well.'

"The Queen finding the wound still so uneasy, sent again to have them open and dress it; but Hulse said it was to no purpose to do anything more and Ranby assured the Princess Caroline he could do nothing that would not give the Queen more pain, without a possibility of doing Her Majesty any good. However, the Queen insisting on having the wound cleaned at least, the King who had told the Queen all that the surgeons had told him, came out, called in Ranby and Hulse and made them comply with her request. As soon as the surgeons had applied some of their lenient ointments and anodyne preparations they left the room, came to Lord Hervey and confirmed their former report of the impossibility of her holding out many hours.

"She then took a ruby ring off her finger, which the King had given her at her coronation and, putting it upon his said; 'This is the last thing I have to give you—naked I came to you and naked I go from you. I had everything I ever possessed from you and to you whatever I have I return.'

"It is not necessary to examine whether the Queen's reasoning was good or bad in wishing the King, in case she died, should marry again. It is certain she did wish it, had often said so when he was present and when he was not present and when she was in health, and gave it now as her advice to him when she was dying; upon which his sobs began to rise and his tears to fall with double vehemence. Whilst in the midst of this passion, wiping his eyes and sobbing between every word with much ado he got out this answer; 'Non—j'aurai—des—maîtresses.' To which the Queen made no

other reply than; 'Ah! mon Dieu! cela n'empêche pas.' I know this episode will hardly be credited but it is literally true. When she had finished all she had to say she said she fancied she could sleep. The King said many kind things to her and kissed her face and her hands a hundred times but even at this time, on her asking for her watch, which hung by the chimney in order to give it him to take care of her seal, the natural brusquerie of his temper, even in these moments, broke out which showed how addicted he was to snapping without being angry and that he was often capable of using those worst whom he loved best. For on this proposal of giving him the watch to take care of the seal with the Queen's arms, in the midst of sobs and tears he raised and quickened his voice and said 'Ah; my God! let it alone; the Queen has always such strange fancies. Who should meddle with your seal? Is it not as safe as in my pocket?'

"The Queen after this fell into a sort of dozing, during which time the King often said; 'She is dying; she will go away in this sleep; it is over; she will suffer no more.' However when she waked she said she found herself refreshed and much better, adding; 'I know it is only a reprieve to make me suffer longer, and therefore, I wish it was at an end; for I cannot recover; but my nasty heart will not break yet.'"

(*To be continued.*)

THE OPERATION FOR CLEFT PALATE.

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The sufferer from cleft palate has one great disability; he is unable to shut off the nose from the mouth at will. The object of operation must be the production of this missing power, and it is a reasonable assumption that the simplest way to produce it is to imitate the normal mechanism. Consequently the first step in planning the operation must be to define the exact manner in which the closing of the nasopharynx is normally performed. It is surprising, however, to see how little attention is given to this point in standard works on cleft palate. Usually the operation is treated almost as if it resembled that for hare-lip in having for its object appearance rather than function. In spite of the soft palate's being composed of actively contractile muscle, it seems to be assumed that it closes the nasopharyngeal passage by falling passively against the posterior wall of the pharynx, in the manner of a flap valve. Arbuthnot Lane, for example, amid almost universal approval, taught an operation which consisted in forming a soft palate simply of two layers of mucosa, not at all unlike a cardiac valve in structure. Similarly, in books of anatomy and physiology the action of the muscles of the palate and the nasopharynx is described in the vaguest and briefest way, with no attempt to show what actual use to the body their contractions serve. In this paper an attempt is made to give an admittedly incomplete account of the action of the normal soft palate, and from this and the principles of general surgery to deduce a technique of operation. Whatever originality emerges in this process is mainly in the arrangement of well-known facts, and in the conclusions drawn from them.

MECHANISM OF THE SHUTTING OFF OF THE NOSE FROM THE MOUTH.

I am convinced that the mechanism which closes the nasopharynx is the same as that which closes so many other passages in the body—the device of a complete muscular ring or sphincter (*Fig.* 1). This ring is set in a plane running backwards and slightly upwards from the middle of the soft palate to the posterior pharyngeal wall, just below the adenoid bed, at the level of the foramen magnum. It is not a simple circlet like the sphincters of the bowel, but is composed of many bands of muscle, interlacing and decussating with each other in the manner familiar to anyone who has dissected this region.

The complicated formation of this sphincter can best be understood if it is separated into anterior and posterior halves (*Fig.* 2). Each half consists

of a muscular sling, and each sling consists of two pairs of muscles, interacting with each other in curious ways.

The posterior sling is made up of a specialized band of the superior constrictor, and of the fibres of the palatopharyngeus which spread out over it. These two muscles by a simultaneous contraction produce a

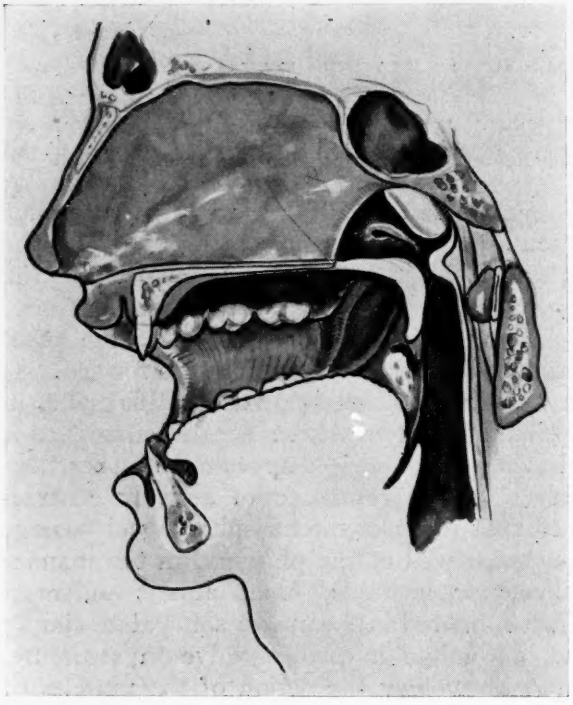

FIG. 1.—Median section of the head, showing the way in which the nasopharyngeal sphincter shuts off the nose from the mouth in saying 'Ah.' Passavant's ridge is projecting forward to meet the posterior surface of the soft palate where it is dragged back by the levatores palati. The uvula is curled forward, and the posterior edge of the palate is not in contact with the back wall of the pharynx.

shelf on the posterior wall of the pharynx which is well known as Passavant's ridge,[8] though of course it is simply the posterior half of the septum produced by the action of the sphincter. The formation of this ridge is peculiar, as it is partly due to the active contraction of the section of the superior constrictor which forms it, and partly to the

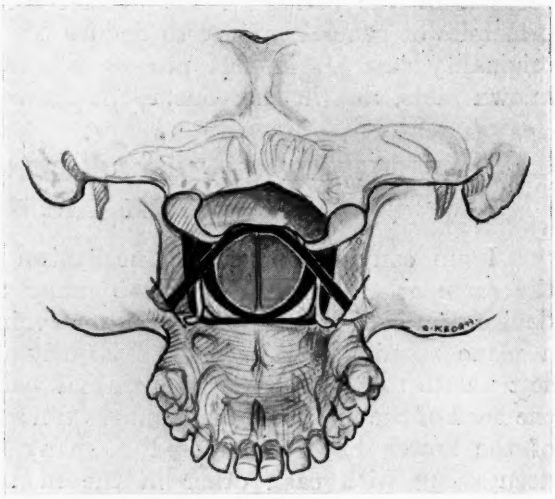

FIG. 2.—Diagrammatic drawing of the base of the skull, showing the muscles forming the sphincter (in solid black). This figure is the foundation for the series of diagrams in *Fig.* 6. It shows the formation of the two slings, and omits the palatopharnygeus as complicating the picture. There is, of course, a certain amount of distortion in order to force a scheme of three dimensions into the limits of two; but if anyone can design a better diagram I shall be only too willing to adopt it.

passive folding of this section into a ruck, by the lifting action of the palatopharyngei (*Fig.* 3). Their action can be illustrated by lifting a trouser leg, representing the constrictor,

by the two fingers and thumbs on either side, representing the palato-pharyngei. As the cloth is pulled upwards it falls into a ruck, but in the case of the pharynx this ruck itself contracts actively at the same time as it is lifted.

The anterior sling consists of the two levatores and the two tensores of the palate. The action of the levatores is the obvious one of lifting and dragging back the centre of the palate against the posterior half of the sphincter; but the tensores, instead of helping this action as the palato-pharyngeus helps the action of the superior constrictor, act in definite opposition to it. This is because of the change in direction given to their tendons by their turn round the hamulus of the pterygoid, so that, though the bellies of tensors and levators are so closely alike in origin and direction, the tensor pulls the palate down against the upward pull of the levator. The result is

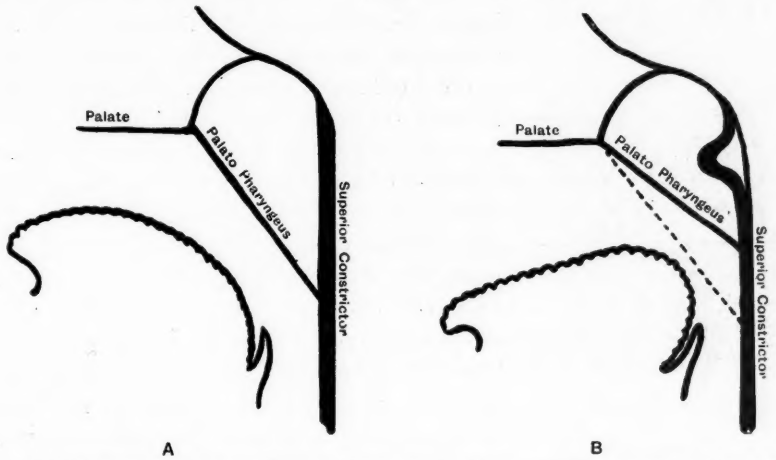

FIG. 3.—Diagram of the formation of Passavant's ridge. A, The posterior sling relaxed, with the constrictor flat against the vertebræ behind, and the palatopharyngeus at full length; B, The way in which the contraction of the palatopharyngeus pulls up the constrictor into a ruck on the posterior pharyngeal wall.

the same as with any other opposing pair of muscles in the body, for instance, the biceps and triceps of the arm: the structure under their control can be kept rigid in any desired position. The importance of this for voice production is obvious. It is interesting to note that this theory of the action of the muscles is borne out by their nerve-supply. The tensores palati are supplied by the otic ganglion, while the muscles which blend into the sphincter are all innervated by the pharyngeal plexus.

Once the sphincter thus described is recognized, it becomes obvious. It can be seen in action by a throat mirror, or felt to grip the examining finger like the sphincter ani. One of the best demonstrations of its action is to push a small rubber catheter through the nose down into the throat, and with this in position to swallow. As the sphincter contracts it rises, owing to the action of the palatopharyngeus already described, and the consequence is that the catheter is gripped and forced out of the nose for a short distance every time the nose is shut off from the mouth in the act of swallowing.

In speech the action of the sphincter is much lighter than in swallowing or gagging, but if the lower end of the catheter be watched through the mouth during the saying of 'Ah', it will be seen to move upwards and towards the centre line. The mechanism is the same in both instances, but the degree of action is very different. Compare the difference between the light touch of the lips in talking, and their forcible screwing up to prevent the entrance of a tongue-depressor.

It is curious, however, to see the disbelief with which a diagrammatic drawing of the sphincter (*see Fig.* 1) is greeted by those used to the old conception of the action of the palate. And yet it merely consists of the co-ordination of two easily observed actions of the body. The first of these is the formation of Passavant's ridge, which is best seen when a cleft-palate case gags and the constrictor sling leaps forwards at each spasm (*Fig.* 4). The second is the action of the palate when the nose is shut off from the open mouth in saying 'Ah.' It will be seen that the middle of the palate goes back into a sort of dimple with the contraction of the levatores, while the posterior edge does not touch the back of the pharynx, as it should if it were acting as a flap valve. On the contrary, the azygos uvulæ, acting in concert with the other muscles of the same innervation, curls the posterior edge of the palate forwards. Thus it is not necessary for the proper action of the soft palate that it should be long enough for its edge to reach the posterior wall of the pharynx, as is so often stated. Perfect control of the nasopharyngeal opening, and consequently perfect speech, is possessed by many in whom, owing to congenital defects, or to surgical attacks on the tonsil and uvula, the palate is so short that its edge can never meet the back wall.

FIG. 4.—Drawing of the preliminary operation showing the appearance of Passavant's ridge when the patient is gagging after the removal of the tonsils, whose fossæ are seen to be empty. The incision for dividing the posterior palatine artery is shown on the left.

If the foregoing account of the action of the soft palate is accepted, the aim of the operation obviously becomes the construction of a contractile ring, as near as possible in structure to the normal one, and capable of closing the nasopharyngeal passage. In order to be effective this sphincter must be continuous in front with an air-tight septum between the nose and the mouth. In clefts involving the soft palate alone this septum is of course the natural hard palate, but in complete clefts an artificial junction of the sides has to be accomplished in this region too.

The conception of a sphincter as the final result of the operation at once eliminates flap operations of the Davies-Colley and Lane variety. It also explains their notorious failure to give normal speech by means of the thin layers of epithelium and scar tissue which they produce. Similarly, the recollection of the proper function of the anterior sling should

prevent a thin atrophic soft palate, containing hardly any muscle (*see Fig. 6 e*) from being called, as it so often is, a 'perfect anatomical result' of operation.

SOME ANATOMICAL CONSIDERATIONS.

It is, of course, impossible to give a formal description of the anatomy of cleft palate within the compass of this article. The following observations merely emphasize certain points which have a practical bearing on the performance and results of the operation.

1. **Increase of Surface and Decrease of Tissues in the Deformed Parts.**—The normal palate, like other parts of the body, has a definite proportion between the area of its epithelial surface and the mass of the tissues beneath. In cleft palate, as in the similar condition of hare-lip, there is a most important change in this proportion; the area of the epithelium is increased and the mass of the tissues is diminished. The surface is increased because the rudimentary epithelium covering the edges of the palatine processes, which should have been absorbed when they met and fused in early fœtal life to form the palate, has survived to develop into normal membrane. The tissues beneath, on the other hand, are lacking in bulk, partly from original failure to develop, and partly from the lack of that stimulus to growth which only use can give. This lack of bulk is for some reason dogmatically denied by certain writers, but there is never enough tissue present to make a palate of normal fullness, even though one of normal function can usually be produced.

The aim of operation has already been defined as the creation of a soft palate as nearly as possible resembling the normal one in structure, and consequently in function. One of the most important elements in the resemblance is the proportion between the surface area and the mass of tissues it covers. In consequence it is not only permissible, but it is imperative, to remove large amounts of mucosa from the surfaces to be joined. The omission to do this is one of the commonest causes of a result like that in *Fig. 6 e*; in which the soft palate consists mainly of mucosa which should have been removed at operation.

This disproportion is, of course, the exact opposite of that which is found in plastic operation for injuries. In these the problem is to restore the loss of epithelium from a normal or nearly normal amount of tissues beneath. The distinction is a very important one, and failure to draw it has been responsible for a good deal of loose reasoning. In cleft palate, as in hare-lip, to preserve every scrap of surface is to ensure a bad result.

2. **Formation of the Bony Boundaries of the Cleft.**—It is strenuously maintained by Brophy that in cleft palate there is invariably a widening of the alveolar arch beyond the normal. A widening certainly exists at birth in some cases, though not by any means in all. Even without operation this widening disappears as the child grows, and apparently no adult skulls can be found to show it. The widest clefts of the palate are associated with wide clefts of the lip, and the joining of the lip in infancy has a very marked effect in narrowing the alveolar arch. Brophy's belief justified his well-known method of pulling the alveolar ridges together in the first months of life, but

apart from many other objections it seems to me that the sacrifice of the germs of the permanent teeth by leaving septic wires among them for long periods is too high a price to pay for an easier joining of the palate.

3. Blood- and Nerve-supply of the Palate. — The blood-vessels and nerves of the palate can be described together in an unsystematic but useful manner as falling into three main groups.

The first group is the posterior one, supplying the soft palate. The blood-vessels in it are several small branches from the external carotid. Its nerves are the motor branches to the muscles from the otic ganglion and the pharyngeal plexus, and the sensory twigs to the mucosa from the glossopharyngeal nerve. From the operator's point of view the most important fact about this group is that none of its constituents is in any danger of injury in the procedures recommended in this paper.

Secondly there is the middle group. This consists of the posterior palatine artery and the sensory nerves to the hard palate which accompany it. In contrast to the posterior structures, these cannot be ignored in the operation. They tether down the mucoperiosteum of the hard palate, just at the point where most mobility is required, by their emergence from the bone at the posterior palatine foramen.

The posterior and the middle group remain the same whatever the degree of deformity, but the third group, consisting of the branches of the nasopalatine artery and nerve which enter through the incisive foramen, varies according to whether the cleft involves their point of entry or not. If the septum is separated from the hard palate by a complete cleft, it is of course impossible for either artery or nerve to reach it. This is, however, of no practical importance; the anterior end of the posterior palatine artery must always have some anastomosis with a similar terminal twig of one of the many branches of the internal maxillary, which give this region so rich a blood-supply. In consequence there is always available an anterior source for the collateral circulation to be described later.

4. The Point of Danger.—It is well known that breakdown of the line of junction after operation almost invariably starts at the junction of the hard and soft palates. The reasons for this being the point of danger lie in the anatomy of the parts, and may be described under four heads:—

a. The clefts that are the most difficult to close (the complete or nearly complete ones) are at their widest at this point. In front the sides always slope together till the alveolar ridge is reached, and posteriorly the halves of the uvula can always be made to meet with ease.

b. There is in this region a sudden change in the substance and shape of the tissues to be joined. The thin, tough, and immobile mucoperiosteum of the hard palate leads without graduation of any sort into the thick, soft, and actively contractile muscle behind. This change calls for different methods of joining the different tissues, and for special care at the point of transition.

c. The three strongest anchorages of the flaps to be joined are all concentrated here. The actual line of junction between the hard and soft palates is marked by the insertion of the palatine aponeurosis into the posterior edge

of the bony palate. In front of this there is the posterior palatine artery, and behind it the tendon of the tensor palati.

d. As the muscles of the soft palate pull backwards as well as upwards on the line of healing, any split due to their action will begin at the anterior border of the muscular ring rather than at the posterior one.

5. The Line of Division between the Muscles of the Sphincter and those External to it.—This line of division is the pterygo-mandibular raphé, which has the sling of the superior constrictor arising from it on its inner and posterior side, and the buccinator taking origin from it anteriorly and externally. As might be expected from analogy with other regions of the body, no important vessels or nerves cross this raphé, and an incision along its line to detach the sphincter from the structures to its outer side can be made with a minimum of damage.

SOME GENERAL PRINCIPLES OF OPERATION.

The tissues to be moved, and the functions to be performed by them in their final position, differ so widely in the two divisions of the palate that these divisions are best discussed separately. In the hard palate the sole object is the making of a septum, much as in an operation for the cure of a vesico-vaginal fistula. In the soft palate, however, there is the very different problem of moving muscles in order to give them a definite function in their new position, and many of the rules governing this type of work elsewhere have to be observed. It must never be forgotten that the production of a simple stiff partition between the nose and the mouth, which means success in dealing with the hard palate, means failure when dealing with the soft.

The Hard Palate.—

1. *The Closing of the Gap.*—The mucoperiosteum of the hard palate is practically unstretchable, so that it must be shifted in one way or another to cover the central cleft. In the classical Langenbeck operation the mucoperiosteum on either side is detached from the underlying bone to form two flaps, attached along their outer margins to the alveolar ridges. These flaps are then swung downwards till their inner margins meet in the mid-line like the sides of a double door. The amount of freedom gained by this swinging on the hinge of their attachment to the alveolar margin is strictly limited by the width of the flap and the inclination of the palatine process on which it lay, so that in many cases, especially those with flat arches to the roof of the mouth, it is insufficient to give easy joining in the mid-line (*Fig.* 5 *b*).

If, on the other hand, the mucoperiosteum is boldly detached from the alveolar ridge along its outer border so that it is simply left attached by its anterior and posterior ends, it can be pulled inwards to any extent needed by the width of the cleft, and still left in contact with the underlying bone. For reasons to be given later this keeping of contact with the bone is of great importance (*Fig.* 5 *d*).

2. *The Gaining of a Wide Area of Contact between the Flaps.*—It is obvious that the wider the area of contact between the raw surfaces of the flaps, the better the chance they have of joining together rapidly and firmly. The simple edge-to-edge junction of the Langenbeck method has a comparatively

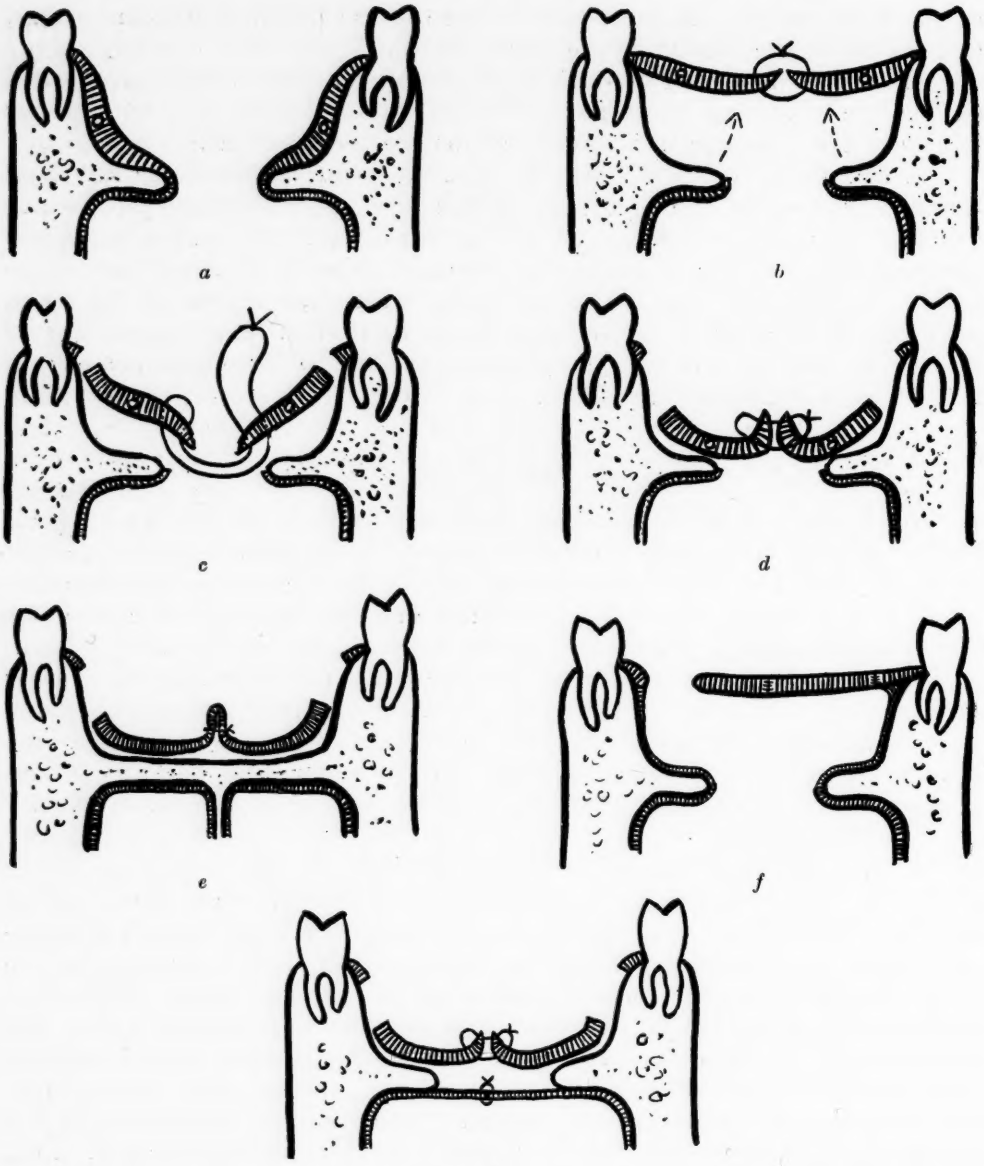

FIG. 5.—Diagram showing the treatment of the hard palate. (*a*) Untouched cleft of hard palate. The septum is ignored for the sake of simplicity. (*b*) Cleft closed by Langenbeck's method. The mucoperiosteum has been swung out from the bone on the hinge of its attachment to the alveolar ridge, till the edges of the cleft meet. The upper surfaces of the mucoperiosteal flaps are left bare, to granulate and contract. (*c*) Recommended method of treating the mucoperiosteum. It has been detached from the alveolar ridge as well as from the palatal process, and a vertical mattress stitch has been inserted. (*d*) Result of tightening the suture inserted in the previous diagram. The edges of the cleft have been everted against each other, and the mucoperiosteum will heal firmly to the bone in its new position. The raw surfaces left laterally are of no importance, as they have a floor of bone. The dangerous area of raw surface is confined to the actual width of the cleft in the bone. (*e*) Method of raising a ruck in the thin mucosa above the apex of an incomplete cleft. (*f*) Formation of a lateral fistula, owing to insufficient mobilization of the flaps, which has not allowed the joined mucoperiosteum to settle against the bone. (*g*) Diagram to show the mucosa lining the floor of the nose joined by Veau's method, as an addition to the closure of the roof of the mouth shown in (*d*).

poor expectation of junction; but if a strip on either side at least 5 mm. wide can be everted against its opposite flap, healing is almost certain. This eversion can be quite easily obtained by means of a vertical mattress stitch, provided that the flaps are sufficiently freed.

When the cleft does not extend throughout the hard palate, it is necessary, in order to gain this eversion at its apex, to insert a couple of mattress sutures in the intact mucoperiosteum in front of it (*see Fig.* 7) in order to raise a ruck there in the mid-line. This means that instead of having an edge-to-edge contact of the thin tissues that normally form the apex of the cleft, there is a wide junction. It is a simple trick, but an almost infallible guarantee of success in the treatment of an incomplete cleft (*Fig.* 5 *e*).

3. *The Maintenance of the Blood-supply to the Healing Surfaces.*—Healing in any part part of the body varies in certainty and speed with the abundance of the blood-supply. It has already been stated that free mobility of the lateral flaps is essential, and also that the posterior end of the mucoperiosteum of the hard palate is tethered to the bone by the structures emerging from the posterior palatine foramen. If these are left intact, they anchor the flaps at just the point where mobility is invariably most needed; yet if they are cut at the time of operation, there is both free bleeding from the severed artery, and, what is more important still, a great diminution of blood to the healing surfaces. The solution of this dilemma appears to me to be the deliberate arrangement of an adventitious circulation to replace the natural one, by the cutting of the posterior palatine artery at a preliminary operation. This means the destruction of the middle artery of supply to the palate, and the taking over of its functions by the natural enlargement of its anastomoses at either end. It is a manœuvre strictly in accordance with the changes of circulation deliberately arranged in plastic surgery elsewhere—for instance, in caterpillar flaps.

The unavoidable cutting of the sensory nerves at the same time as the artery I regard as of no real importance. They must regenerate in time in most cases, and I have never seen the slightest trouble from their section.

4. *The Removal of the Strain of Junction from the Soft Palate.*—However free the mobilization of the sides of the cleft may be, there must remain an irreducible minimum of tension to be overcome in holding them together. This tension should be put as much as possible upon the tough and unimportant tissues of the hard palate, rather than upon those flimsy muscles of the soft palate which determine normal speech. If the hard palate is dealt with in the way recommended, it will be found that the sides of the gap in the soft palate are almost or quite in contact, and need only light co-aptation sutures to hold them together.

5. *The Minimizing of Post-operative Contracture.*—Wherever an area of the body is left denuded of epithelium, that area tends in the process of healing to contract forcibly. It is contraction of this type which causes the narrowing of the alveolar arch which is so common after the joining of a cleft of the hard palate, and which also fixes and stiffens the soft palate.

In the Langenbeck operation the raw area left is immense—the entire width of the alveolar arch in many cases—and there has been very little discussion as to how exactly this heals. Raw areas must be left in any

conceivable operation, but there is a very important distinction to be drawn between two different types of these. In the first the raw area is an exposed surface of bone, the rigidity of which is quite capable of resisting the contracting force exercised upon it in the process of healing. The gaps left at the outer edges of the hard palate flaps are of this sort, and in consequence cause no contracture and are entirely harmless. In the second type the raw surface is unsupported by bone, and exerts its full pull upon the structures at its margins. It is this kind of healing area which is left upon the upper surface of the mucoperiosteum after Langenbeck's operation, and its contraction bends the bony arch above as a bow-string bends a bow. In addition to this action it must drag the upper surface of the soft palate backwards and downwards, fixing it in the worst possible position for fulfilling its function. If the hard palate flaps are slid inwards in the way recommended, the raw surface of the dangerous type left is no more than the area of the actual bony gap, instead of extending from one alveolar ridge to the other. By adapting the procedure of Veau (*Fig. 5g*), and making a complete lining to the floor of the nose from the mucosa of the palatal process and the vomer, it should be possible to reduce this dangerous raw area almost to nothing. I have had insufficient experience of Veau's technique to judge whether the making of this inner nasal lining is worth while, but in complete clefts it seems a most reasonable proceeding, and there is no great difficulty in adding it to the joining of the oral surface.

The lateral fistula that is sometimes described as persisting after the making of relaxation incisions in the hard palate is the result of half measures —that is to say, the freeing of the flaps has only been enough to let their edges meet in the middle, and not enough to allow them to fall against the bone (*Fig. 5f*). In consequence the inner side of the lateral incision is held stretched away from the bone till epithelium grows over it in this position. If the flaps are properly mobilized this cannot occur.

The Soft Palate.—

1. *The Closing of the Gap.*—The simplest method of closure, that of simply sewing together raw surfaces produced on either edge of the cleft, fails in such a large number of cases that many procedures have been devised for combating the tension which is the most obvious cause of breakdown.

Fergusson,[5] in a pre-anæsthetic and pre-antiseptic era, obtained a remarkable percentage of joins, in incomplete clefts, by cutting the actual palate muscles on either side. It is hardly necessary to point out the disadvantages of this Gordian solution of the problem.

Arbuthnot Lane[7] ignored the muscles completely, and closed the gap with a flap of mucosa. If voluntary closure was ever possible after this, it could only be by an abnormal development of the posterior sling.

Brophy[4] and Veau[9] rely on secondary tension sutures of wire which stretch the muscles to such an extent and for such a time that healing can occur in the mid-line. The pressure and the inevitable sepsis must of course cause considerable scarring and necrosis in the very tissues on which speech depends, but both surgeons recommend these sutures as avoiding the damage done by relaxation incisions (*Fig. 6d*).

Gillies,[5] with a welcome recognition of the importance of function rather than appearance, has suggested detaching the soft palate entirely from the hard, and suturing it separately. However, as perfect function can admittedly often be attained combined with complete closure, it seems a counsel

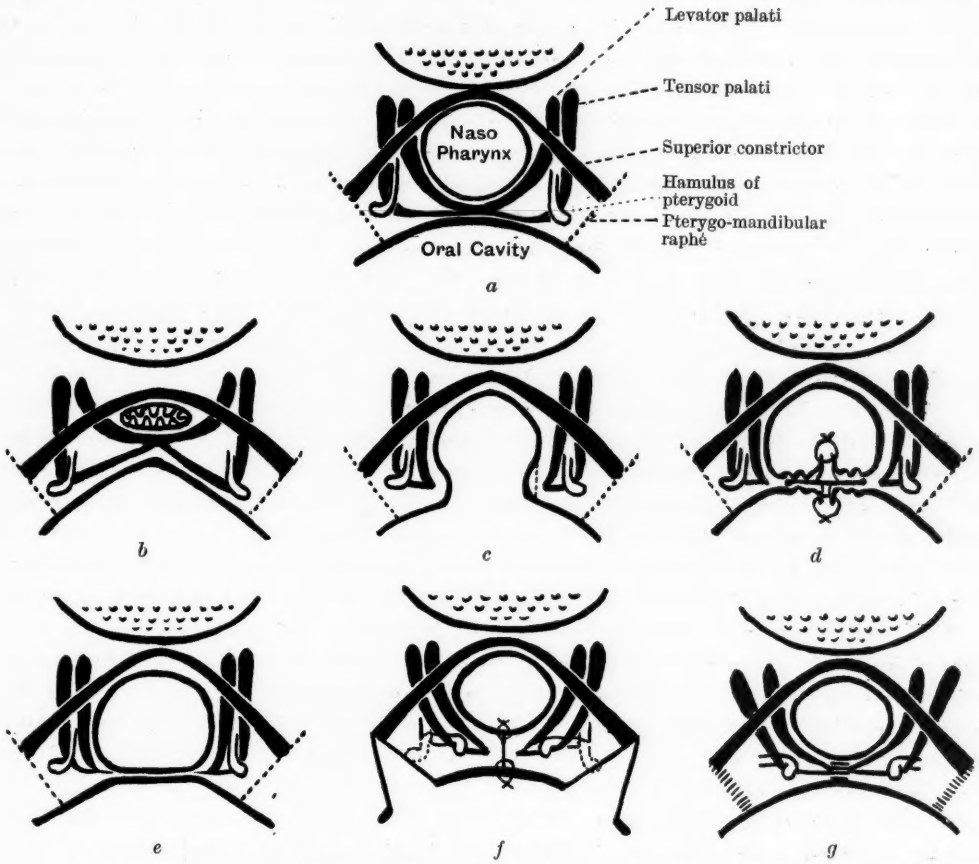

FIG. 6.—Diagram showing the treatment of the soft palate (*see Fig.* 2). (*a*) Normal sphincter in relaxation. The constrictor and the levator palati are relaxed, and the tensor is contracted, pulling the soft palate away from the posterior pharyngeal wall. (*b*) Normal sphincter in contraction. The tensor is relaxed, allowing the middle of the soft palate to go up into a dimple. The constrictor and the levator are contracted, shutting the nasopharyngeal passage. (*c*) Formation of a cleft of the soft palate. The bony framework is the same as before, but both the muscles of the anterior sling are shortened, and end not far from the hamular process. The line of section of the mucosa advised is shown on the right. (*d*) Method of joining palate by splitting the edges of the cleft and suturing them, while the strain is taken by a submucous suture of the type of Veau's. The results of other types of relaxation sutures can be easily substituted. (*e*) The result usually produced by suturing without shifting the muscles. The palate is thin and tight, and rather farther away from the posterior wall than normal. (*f*) Method of suturing recommended. Relaxation incisions have been made along the pterygo-mandibular raphé, and the hamulus has been snapped off inwards. This allows the ends of the muscles to meet without stretching. A wide strip of mucosa has been removed, and the raw surfaces are joined by two rows of sutures. (*g*) Result aimed at by the operation recommended. The nasopharyngeal passage is narrowed, the ends of the muscles are firmly joined, and the relaxation incisions have healed into a reinforcement of the pterygo-mandibular raphé.

of despair to advocate the payment of the very heavy price of a permanent gap in the palate for the chance of improved speech. I believe that the method recommended in this paper gives even better freeing of the muscles,

combined with better prospects of healing; and it does not condemn the patient to prosthetic appliances that are both difficult to fit and galling to wear.

Finally, the usual text-book of general surgery vaguely advises that "relaxation incisions should be made when closure cannot be otherwise effected", and in its illustrations shows these incisions so placed that their only effect could be to produce dangerous bleeding from the posterior palatine vessels.

It is curious that almost the only solution of the problem that is not advised is to apply to the palate muscles those principles which would be used if they were situated elsewhere in the body. The operation is essentially one of muscle transplantation. The well-known rules of this proceeding are to leave the muscle untouched at its origin, and to free it distally enough to allow an easy reaching of its new insertion, without undue tension. As the purpose of the shifting of the palate muscles is the closing of the gap in a sphincter, this insertion is for each muscle the end of its corresponding one on the other side.

The rigid bony framework of the pharynx is of normal size; the muscles available are short and atrophic. Consequently they need considerable loosening to give an unstrained line of junction, just as the facial muscles need loosening from the underlying framework of the face in closing a cleft lip. The freeing of the levator palati is easy enough, but the turn of the tendon of the tensor palati round the hamular process fixes it firmly to the boundaries of the nasopharynx, as well as changing its direction so that it pulls directly outwards against the line of junction. It is fortunate that the hamular process can be very easily snapped off at its base without interfering with the synovial sheath of the pulley, and so allowed to be displaced inwards and upwards to a position which will not interfere with the joining of the two tensores. In this new position it must finally become fixed by the healing processes so as to afford once more a fulcrum to the tendon that curls round it (*Fig.* 6*f* and *g*).

2. *The Neutralization of Muscular Pull upon the Sutures.*—In the previous section the problem of tension has been regarded as if the tissues concerned were passive structures, whose pull upon the healing line would vary simply with their elasticity and the thoroughness with which they had been mobilized. But there is another kind of tension to be considered as well, the more dangerous and variable active tension due to the contractions of the irritated muscles. These contractions, however, can be robbed of all their danger if the process of joining the gap leaves the distal end of the muscle at a distance from its origin less than its minimum length when contracted. The mobilization recommended does this, and in time the muscles shorten to suit the new conditions, just as the muscles of a limb do when their supporting bone loses length from a fracture.

3. *The Diminution of the Diameter of the Passage to be Controlled by the Sphincter.*—This is another secondary, but very important, effect of the shifting of the palate muscles. It is obvious that if tissues of less than normal length are to be joined into a ring at no more than normal tension, that ring will be proportionately smaller than the normal diameter. *Fig.* 6*f* shows

how the falling upwards and backwards of the line of junction of the gap in the sphincter diminishes the diameter of the passage it has to control, bringing the front half of the sphincter close to the back half (Passavant's ridge). The importance of this in facilitating voluntary closure is easy to understand.

4. *The Provision of a Broad Healing Surface.*—This is as important in the soft palate as in the hard. The best way to gain it is by free removal of mucosa, which, as has already been pointed out, is present in excess. The method so often recommended, of splitting the edges of the sides, and suturing the split halves separately has three disadvantages. First, there is a central dead space left, in which blood-clot can accumulate and nourish infection; secondly, healing will produce a median strip in the soft palate consisting of mucosa alone, instead of a proper junction of the actual muscles; and lastly, the preservation of all the mucosa means that there is a surplus available to line any gap that may appear in the line of suturing, with a consequently greatly decreased tendency of this gap to spontaneous closure.

5. *The Maintenance of the Blood-supply to the Healing Surfaces.*—There is fortunately no danger of cutting the main arteries of supply to the soft palate, as has been already pointed out. It is, however, easy to produce local areas of anæmia by sutures and other devices used to counteract tension, and these areas must suffer from reduced healing power. Fergusson, for instance, who used a suture embracing the entire thickness of the palate, notes that often the only places that failed to join were those directly opposite the stitches. The moral is to reduce this interference to a minimum by employing two layers of fine sutures on the oral and nasal surfaces respectively, which leave between them a broad healing area with a completely free blood-supply. If the mobilization is sufficient there is no need for tension sutures, tapes, and metal devices, which must interfere gravely with the supplying vessels, besides destroying important tissues.

6. *The Minimizing of Post-operative Contracture.*—The comparatively large amount of scar tissue produced at the outer edge of the sphincter by the free mobilization of the muscles does much less damage than a smaller amount in more vital spots. The main mass along the lateral incisions simply reinforces an already existent line of fibrous tissue, the pterygo-mandibular raphé.

As the lateral incisions heal they have a tendency to drag back the displaced tissues into their original positions, but in assessing this tendency it must be remembered that the central suture line has the advantage of healing first, and that the new position of the muscles is confirmed by every contraction they make.

PRELIMINARY OPERATION.

There are two things to be done as a preliminary to the actual joining of the cleft:—

1. The first of these is the rearrangement of the blood-supply by cutting the posterior palatine artery. It is easily done by making a small incision just to the outer side of its emergence from the bone, and then tearing it

through with a dissector. The division of the artery is signalled by a violent gush of blood, which soon ceases under pressure on the wound.

2. The second preliminary is the removal of that forcing bed of infection, the tonsil. I am aware that some followers of Pangloss[2] consider that this organ has a protective function, but personally I believe it belongs to the same order of nuisances as the mastoid cavity, and has as much function as the male breast. It is one of those waste places and weak spots which pervade the human body as they do the human mind. I follow Addison[1] in thinking that the risk of that violent infection of the wound which is the only cause of failure in a properly performed operation is much decreased by previously removing the tonsils. They should be dissected out with the utmost care to preserve every scrap of mucous membrane; there is no excess of surface where they lie. In two of my most difficult cases the guillotine had been used, with the not uncommon result of removing equal proportions of tonsil and soft palate. I do not think the adenoids are ever so dangerous as the tonsils, but if they have accumulations between their folds or appear inflamed they should be removed.

At least three months should be allowed after the preliminary operation for the resolution of all inflammatory products before the main operation is performed.

MAIN OPERATION.

Naturally the child should be free from colds and in as good general health as possible. There is much to be said, in London at any rate, for considering it an operation of the summer season.

I think that the liability to infection of the wounds is undoubtedly diminished by the administration of a prophylactic dose of 5 c.c. of anti-scarlet-fever serum a day or two before operation, as advised by Addison. There is a close connection between the more dangerous throat streptococci and the organism of scarlet fever.

Anæsthetic.—Intratracheal gas and oxygen through a nasal catheter is the best of all, but few are competent to give it. Ether vapour delivered through a weighted self-retaining tube is quite satisfactory. The very greatest care should be taken to keep the airway free and the colour good.

Position.—The patient should lie on a flat table, with the shoulders raised about 3 in. by a sand-bag. This position allows blood to run into the back of the nose rather than down the throat, but the head is not congested as it is if lowered below the main part of the body. The surgeon should sit at the top of the head; thus he sees the structures upside down, as they are illustrated.

Instruments.—I do not think there is any need for the special instruments which fill so many pages of the instrument-makers' catalogues. The problem is that of stitching accurately in a cramped space, and there are few major operations in which it may not arise. The repair of a common bile-duct or of a ureter may expose the limitations of the ordinary needle and needle-holder just as well as the stitching of the nasal surface of the soft palate. Consequently the indication is not to invent special varieties

of these instruments for cleft palates, but to improve the ones in ordinary use till they will pass this or any other test.

The needle-holder I use is one primarily designed for general use, but I have found it to add greatly to the ease of this operation (*Fig.* 7). It has a very wide range of movement for any given position of the hand, it is gripped equally firmly whether open or closed, and the catch can be released by a touch on the trigger without twisting the point, however it is held. To most people control of the jaws by the thumb and release of the catch by the forefinger come automatically.

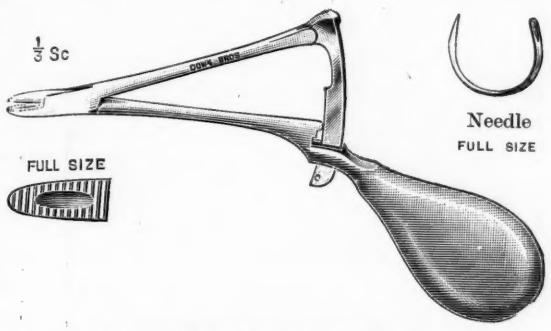

Fig. 7.—Needle-holder and needle described in the text. The needle is more slender than it appears in the illustration.

The needle is 25 mm. in length. It is curved through five-eighths of a circle, so that the point can be inserted into a flat surface and made to reappear after taking a full grip, simply by twisting it. If the curve of the needle is merely half a circle or less it is sometimes difficult to get the point to show after inserting it in the deeper stitches. It has a sharp cutting edge, but this is confined to the extreme end, so that it cannot cut sideways if dragged askew. The ordinary cutting needle with a long edge on either side may do great damage in this way. No other needle is necessary in closing cleft palates, or for that matter in closing hare-lips. Like the needle-holder, it is an instrument of general surgery, with many applications in the under-running of bleeding points and the closure of minute rents. The holes made by it are punctures rather than cuts or tears, and so the tissues lie closely around the sutures, with a consequently diminished liability to infection compared with those that surround the gaps left by the harpoon-like instruments sold as 'cleft-palate needles'.

The sutures should be silkworm gut, which has no capillary attraction for the necessarily infected fluids of the mouth. Fairly thick strands should be used for the hard palate, and finer grades for the soft. There is no need to use the very finest available gut; apart from its tendency to break on tying, it must cut through the tissues more easily than the moderately thick.

An ordinary scalpel, an extremely sharp fine-pointed one, a curved 7-in. Mayo's scissors, a semi-sharp straight dissector, and a fine rat-toothed dissecting forceps complete the outfit needed for the actual operation. In addition a really powerful suction pump to clear away the blood will be found as great a help in this operation as it is in dealing with an appendix abscess or a brain tumour.

Mobilization of the Sides of the Cleft.—The anatomical and technical principles set out above remain the same whatever the extent of the deformity may be. It follows that the operation is much the same whether the cleft is wide or narrow, complete or incomplete. In every case without exception both divisions of the palate, the hard as well as the soft, should be completely

mobilized. This mobilization is obviously necessary when the cleft is complete, but it is also needed in incomplete clefts if the principles of getting broad healing surfaces in contact and allowing unimportant tissues to take the strain of suturing are to be followed.

The lateral incisions follow the lines described by Addison, dividing the mucosa along the whole length of the pterygo-mandibular raphé, and then running forward, close to the inner edges of the teeth, to about the level of the incisor (*Fig.* 8). The soft palate is freed from the buccinator and pterygoid muscles by blunt dissection along its outer side to a depth of a centimetre or more. On the inner wall of this incision the hamulus of the pterygoid can be plainly felt, just as it can be felt through the upper end of the gap left by removal of the tonsil, and it should be snapped off inwards. This can be done by a surprisingly light pressure with the finger nail. It should be remembered that the displacement of the spike of the hamulus leaves palpable its projecting base, which feels very like the original process. The hamulus is never exposed to view, and no damage is done to the synovial sheath of the tendon of the tensor palati as it turns round it. Consequently this muscle can act efficiently again as soon as the hamulus is fixed by adhesions in its new position.

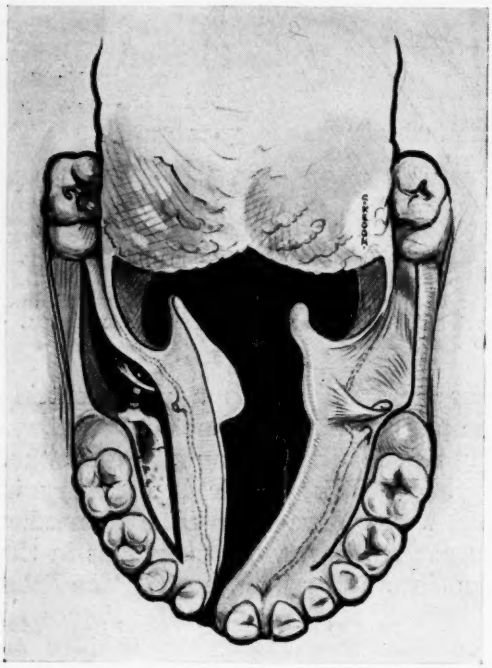

FIG. 8.—Diagrammatic drawing of a complete single cleft. On the right side the line of incision is marked out: the hamulus, the tendon of the tensor, and the posterior palatine artery, are shown as though the mucosa were transparent. On the left side the palate, both hard and soft, has been freed ready for suturing. In the bare palate bone left by its inward shifting can be seen the stump of the artery, which has been cut at a previous operation, while the hamulus is broken off its base and has fallen inwards. Of course the tendon of the tensor palati should not be exposed as it is in the drawing.

Next the mucoperiosteum of the hard palate is levered off the bone by the dissector, avoiding the entrance of the branch of the naso-palatine artery at its anterior end. As the posterior palatine artery has been previously cut, there is no obstacle to the complete and rapid freeing of this flap. Along the margins of the cleft the dissector should be thrust out till it is free in the mid-line, aided if necessary by a touch with the knife.

The last stage in freeing the palate is to separate it by the curved scissors and the dissector from the posterior edge of the bony palate—a proceeding which is made easy by the previous division of the posterior palatine artery. The proof of proper mobilization is that the sides of the cleft tend to fall together, and can be pushed into contact with the very lightest pressure.

Bleeding during this part of the operation is usually free, but no vessel of any size should be divided, and the capillary oozing can be controlled by pressure. It is during this stage especially that a proper sucker will add so much to the ease of the work.

Suturing the Hard Palate.—If the hard palate is cleft, the edges must be dragged together by vertical mattress sutures (*Fig.* 9) of strong silkworm gut till at least 5 mm. of the raw surface on either side is in opposition. At the extreme anterior end of complete clefts it is usually impossible to evert the edges, but a small defect left here usually closes spontaneously.

The procedure is much the same if the hard palate is normal, or only partially cleft. Mattress sutures are used to raise in the middle line the ruck previously described. The making of this fold drags together the edges of the actual cleft behind it, and ensures that at its apex there shall be broad raw surfaces in contact instead of thin edges. I would repeat that if the hard palate is properly treated, the sides of the cleft in the soft part will be almost or quite in contact before any sutures are put in them.

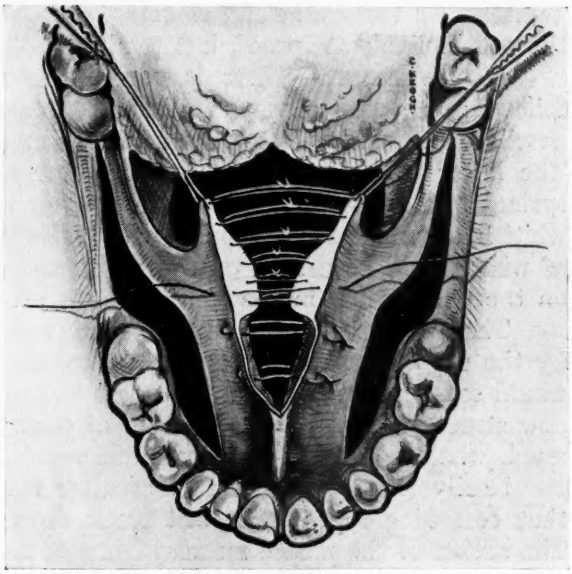

FIG. 9.—Diagrammatic drawing of the stitching of a cleft of the soft and half the hard palate. Both sides have been freely mobilized, and a ruck has been raised at the apex of the cleft by two mattress sutures. Two vertical mattress sutures evert the edges of the muco-periosteum, and the sutures on the nasal surface of the soft palate are shown in place. A single one of the six sutures which will join the oral surface has been inserted.

Suturing the Soft Palate.—The handling of the flimsy edges of the cleft of the soft palate is much helped by passing a fine suture through the tip of each half of the uvula. The ends of these sutures are caught in artery forceps, which are allowed to hang forwards or backwards as is most convenient, to act as steadying weights. Then with the fine-pointed knife a strip of mucosa is carefully sawn off the edge of the cleft, starting the cut by transfixion close to the steadying stitch. This strip should broaden from about 4 mm. near the uvula to about 8 mm. where it meets the anterior raw surface, past the belly of the sphincter. For the reasons given the raw area should err on the side of wideness rather than of narrowness, and the greatest care should be taken to see that all mucous membrane is cleanly removed from it.

Then the nasal surface of the soft palate should be joined by interrupted sutures. The first of these is the most important in the whole operation, and should join firmly and accurately the angles caused by the sudden swell of muscle from the thin mucoperiosteum of the hard palate. It should take

up about a third of the thickness of the raw surface, and the needle should be felt to penetrate the tough layer of the palatal aponeurosis. The succeeding half dozen stitches on this surface take gradually decreasing bites of the tissues, till finally the uvula is formed by fine sutures of the mucosa alone. The insertion of the later stitches will be helped by letting the sutures stretching the sides of the cleft fall forwards over the nose. When this row of sutures has been completed the line of junction should appear as a deep narrow cleft, with straight sides that are in contact for most of their depth. The oral surface is next joined by half a dozen stitches, any dried blood is cleared away, and the operation is complete. I think that the use of Whitehead's varnish or any similar preparation is a mistake; it cannot make the suture line waterproof, but tends to collect and confine the exudation.

After-treatment.—There is very little to be done after operation beyond following the very obvious indications of keeping the child as contented as possible, and getting it to take soft food and an abundance of glucose water. The latter will clean the wound to some extent, and I do not think that syringing the mouth is worth the trouble it almost invariably causes. The whitish accumulations, usually called sloughs, are simply the sodden state of what would be a scab on a dry external wound, and are no more harmful on the palate than in the tonsillar fossa after a tonsillectomy.

That bugbear of the older surgeons, the breaking down of the wound by the use of the child's tongue, never occurs in actual practice; though it would seem natural enough to expect the continual investigation of a totally new structure occurring in the roof of one's mouth. Similarly, I doubt very much whether there is any justification for the use of arm splints.

Really the only thing to fear after the operation described is sepsis of that corroding type which will break down any wound in which it occurs. The action of the palate muscles can put no strain on the suture line for the reasons described. Indeed, I have encouraged several of my more biddable patients to demonstrate the action of the sphincter by talking and saying 'Ah' during the first week after operation, with the stitches still in position. Incidentally it is an interesting psychological point that the use of the sphincter in saying 'Ah' is instinctive, even if it is so incomplete that it can never have shut off the nose.

The stitches on either surface of the soft palate will come away by themselves, but those in the tougher tissues of the hard palate will remain indefinitely if left alone. Consequently they should be removed about the tenth day.

Results.—The free mobilization, neutralization of muscular contraction, and light suturing which have been described give a high percentage of complete unions. In partial clefts failure should be very rare, while in the complete ones it is easy enough to get runs of a dozen successive cases with primary healing of both divisions of the palate.

I would prefer to postpone the discussion of the functional results till a larger number of my patients (most of whom have been operated upon at about 2 or 3 years old) have attained the age of willing co-operation. However, the freedom of movement of palates formed in this way, upon saying 'Ah' or gagging, promises very well.

SUMMARY.

1. The object of the operation is assumed to be the imitation of the normal mechanism of shutting off the nose from the mouth.

2. This mechanism is argued to be a sphincter.

3. In forming this sphincter the ordinary principles of muscle transplantation should be observed.

4. As the muscles are too short to meet without tension they have to be shifted.

5. This shifting allows them to meet, narrows the passage they will have to control, and renders their contractions innocuous to the healing line.

6. This shifting cannot properly be done without changing the blood-supply at a previous operation.

7. Certain forms of raw surface are harmful, and others harmless: the operation is planned accordingly.

8. The tonsil is a danger to the success of the operation, and should be previously removed.

9. The operation is described.

I find it difficult to acknowledge properly what I owe to the privilege of watching the work of my seniors at the Hospital for Sick Children, more particularly Mr. Addison and Mr. Barrington-Ward. As for the illustrations by Dr. C. A. Keogh, there lies in them, if nowhere else, a justification of this paper. Their quality can be appreciated either by comparing them with those which adorn more famous works, or by endeavouring to produce oneself something approaching their simplicity and accuracy.

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A GIANT GALL-STONE IMPACTED IN THE COLON AND CAUSING ACUTE OBSTRUCTION.

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In February, 1931, I was asked by Dr. J. E. Basham, of this city, to see an old gentleman of 81 suffering from acute abdominal pain with vomiting. I had known this patient by sight for nearly forty years and had always recognized him as a stout, healthy-looking man of regular habits and addicted to walking. The family doctor gave me the following history :—

"I was called to attend the patient in February, 1930, for an attack of bronchitis with very troublesome cough. When almost recovered from this illness, he complained of 'pain in the side with a feeling of distension'. The pain was intermittent and there was slight tenderness beneath the left costal margin which did not increase on deep pressure. He was at that time very stout and difficult to examine, and nothing definite was made out. The patient was of a constipated nature, and for years had been accustomed to take a laxative of his own concocting, but in spite of his remedy complained that his bowels had not been moved for two days. After an aperient the discomfort was removed and there was no further disturbance at that time. In July he suffered from retention of urine due to enlarged prostate. The radical operation was carried out, and by the end of September the patient was nearly well again. In November I was again called to see him regarding the pain in the side. The tenderness was more marked, and the pain, whilst not severe, was worse than on the previous occasion ; it was again intermittent in character ; the bowels were not constipated, but the evacuations were very watery and pale in colour. There was no apparent distension of the upper abdomen, but on deep pressure over the tender area the pain was made worse. By means of senna and cascara the bowels were kept open, and gradually the attack subsided. I was certain at this time that he had a chronic intestinal obstruction at the junction of the transverse and descending colon and informed the patient, but he declined further investigation and would not even consent to be X-rayed. No doubt his refusal was due to the fact that he was just recovering from the long illness due to his prostate, and as the attack eased off he wanted to leave things alone for the time being. Early in February, 1931, the patient again suffered from an attack of bronchitis lasting ten days, and just as this was clearing away I was again called to see him on account of recurrence of the pain in the side, the attack resembling that of November, 1930. This was two days before I asked you to visit him. On a Sunday morning he appeared better, but that evening he was much worse, had vomited twice, and there had been no movement of the bowels or passage of flatus. I gave an enema of

¾ pint of hot water, soap, and olive oil with a long tube, with the object of reaching the area where I thought the obstruction was situated. After inserting about two to three feet of tube I felt the end strike against something very hard, and on gently pumping the syringe no fluid would go through. I withdrew the tube an inch or two and tried again; this time the solution flowed without obstruction and the rubber tube could then be passed farther than where it had previously struck the hard substance. The patient obtained instant relief, which continued until the next morning, when he became worse and I called you in."

When I saw this old gentleman he was in the throes of an attack of intestinal obstruction and looked very ill. The abdomen was much distended and the contracting cæcum could be both seen and felt. The diagnosis appeared obvious enough, and I had no doubt that he was suffering from a malignant growth about the splenic flexure with a superadded acute obstruction. With as little delay as possible he was removed to a private hospital. Using local anæsthesia I exposed and opened the cæcum, with immediate and complete relief (this was on Feb. 16). His general condition rapidly improved and he began to take nourishment. Once or twice he felt as though the bowels might move, but there was only a small evacuation and he passed no flatus by that route. After the distension went down, a large mass could be felt in the left hypochondrium and this was looked upon as undoubtedly a new growth in the large bowel. During succeeding days it was observed that this mass diminished in size. As there was no evidence of dissemination it was decided to explore the abdomen without further investigation, for it was quite clear that in any event he could not be left with an open cæcostomy, and if, as I feared, the growth could not be removed it was my intention to make a lateral anastomosis and to close the cæcum.

On March 9 the abdomen was opened by a vertical incision in the vicinity of the mass; the abdomen was bulky, and, rather than expose the mass in its entirety, it was explored by palpation. It was large, rounded, and intensely hard, and I was in considerable doubt as to its nature. Still thinking that we had to deal with a growth, the liver was examined, but there were no secondary deposits. Incidentally the gall-bladder region was palpated and was found to be surrounded by what appeared to be a mass of adherent bowel and omentum. The incision was now enlarged to enable me to examine the mass more thoroughly and perchance to withdraw it from the abdomen for inspection. It was then apparent that there was something in the transverse colon just like a large bobbin, but it was so enormous that I thought more of an enterolith than of a gall-stone. Whatever its nature, the wall of the bowel was so tightly stretched over it that it was quite anæmic and the longitudinal bands were so spread out as to be merged and indistinguishable. The mass could not be moved, so an incision was made directly over it in the long axis of the gut. The moment the bowel was opened there was no longer any doubt as to its nature, for I saw that we had to deal with a gall-stone of unusual size. It required an incision about 2½ in. long to enable the calculus to be extracted without tearing the intestinal wall. On removal, it was noted to have a terminal facet, and this prompted a further search, when a second,

large enough, but much smaller, calculus was found lying in the bowel on its proximal side, and this was removed. There was very little fæcal escape, but what little muco-fæcal discharge occurred was exceedingly foul, as indeed were the calculi themselves.

Owing to the emptiness of the bowel the bed in which the gall-stone had lain could be carefully inspected. It was not ulcerated, but was mottled with curious pink areas which looked like a cutaneous surface and may have represented healed ulcers. The intervening mucous membrane was dark purple and with an appearance like the pile on velvet. The incised bowel wall was sodden and friable and in consequence a little difficult to suture. As the lumen was in no way narrowed, the incision was closed in the longitudinal direction, being fortified externally by neighbouring omentum. This operation was carried out under general anæsthesia and was followed by some shock, but the patient soon rallied, and in a few days passed some flatus per anum, though almost the whole of the intestinal contents continued to escape by the cæcostomy. About ten days later he was not so well, his appetite failed, and he developed symptoms of mental derangement amounting to delusional suspicion. On March 23 the cæcostomy was closed ; this interference again necessitated a general anæsthesia as the surrounding skin was so sore.

For a time the mental confusion improved, but by the end of two weeks it was so troublesome that as a therapeutic measure the patient was sent back to his home surroundings. There he soon improved, and by the end of April was able to be up and about, and has since been restored to his normal health. In the light of the disclosures of the operation, this patient was very closely questioned and was encouraged to search his memory, but he could not recollect ever having suffered from abdominal pain until the attack twelve months previously.

The calculi removed are certainly noble specimens. The larger stone when fresh weighed 5 oz. (4800 gr.) and measured 3 by $2\frac{1}{4}$ by 7 in. circumferentially ; even the smaller stone turned the scale at 1 oz. (960 gr.) and measured $1\frac{3}{4}$ by $1\frac{1}{2}$ by $4\frac{1}{2}$ in. circumferentially (*Fig.* 10). It is of interest to note that the calculi lost in weight as they dried and that their surfaces began to crack and peel. The illustration shows the exact size of the stones, but somehow they do not look either large enough or sufficiently massive. I suppose that only stereoscopically would they appear at their best, but even then, compared with their startling size when first removed, it would be as freshly caught salmon shimmering on the gravel by the river side to the same fish weltering on the marble slab of the shopkeeper ! Both calculi have found a fitting asylum in the Royal College of Surgeons Museum, but there they must make their neighbours feel at a disadvantage, for even in that galaxy of giants there are none that can approach them in magnitude. A search of the British literature, willingly made by Mr. S. Wood of the Royal College of Surgeons, did not unearth the record of any calculus approaching the larger stone in size, the nearest by weight being only 3 oz. and 5 drachms. Envious critics stoutly asserted that so large a concretion must be an enterolith, but Mr. T. W. P. Lawrence has kindly roughly assayed the stone for me, and finds it composed almost entirely of cholesterin and bilirubin pigments. He has no doubt but that it belongs to the genus Gall-stone.

In all these cases the question of the route by which such calculi enter the intestine always arises. I discussed this matter in a paper on " Intestinal Obstruction from Gall-stone " in 1927.[1] The point is so constantly raised

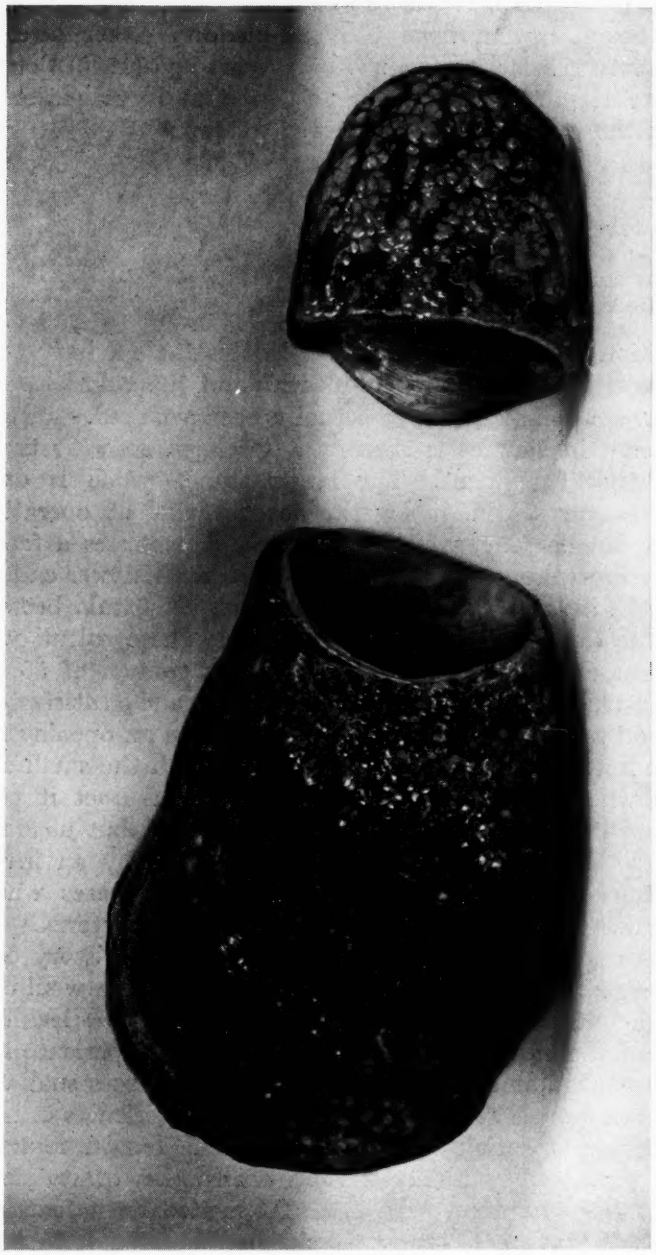

FIG. 10.—The calculi. The larger weighed 5 oz., and measured 7 in. in circumference. (*Actual size.*)

and is so interesting that I crave the permission of the editor and the indulgence of the reader for the repetition of the following paragraphs from that paper.

"The absence of previous symptoms of gall-stones is often a remarkable feature. When confronted with these cases people invariably say what a curious thing that the patient has never had a history of jaundice! As a matter of fact, gall-stones which are sufficiently large to produce intestinal obstruction do not often traverse the common duct, but find their way into the intestine by ulceration from the gall-bladder, either directly into the duodenum, or into the large bowel. Many years ago (1890) Courvoisier went carefully into this matter, and from post-mortem investigation of 36 cases he found that the common routes were as follows:—

Through the gall-bladder—
Duodenal fistula twenty-five times.
Ileum fistula once.
Colon fistula once.
Duodenal and colon fistula twice.
Through the common bile-duct seven times.

"In addition to the routes specified in this table I have seen the communication directly between the gall-bladder and the stomach, and cases are on record in which gall-stones have actually been vomited. In the College of Medicine Museum in Newcastle there is a specimen showing two gall-stones, composed mostly of pigment. The larger stone, which is dark green in colour, and measures 2½ in. in length, was removed at operation from the stomach. The smaller one, which is brown and possesses a facet which fits the end of the larger stone, was found impacted at the lower end of the ileum, where it was the cause of intestinal obstruction. A fistula between the gall-bladder and the duodenum was demonstrated. These calculi were removed by my colleague, Mr. T. A. Hindmarsh, from a woman of 53 on whom he operated for obstructive symptoms. The patient made a satisfactory recovery, and was in good health a year later. There may be an opening into all three neighbouring viscera, and not uncommonly into both the small and the large bowel. Even when the stone traverses the common duct it probably does not usually find its way through the normal opening, but ulcerates from the ampulla directly into the bowel, and this is proved by an investigation of museum specimens. Among great rarities there are cases where the gall-bladder has burst into the urinary bladder or even the uterus. A knowledge of pathology, therefore, does not lead one to expect a history of jaundice in many of the cases, but it might be supposed that there would always be a history of a definite and perhaps severe gall-stone illness leading up to the passage of the stone into the bowel. As a matter of experience this is not by any means always so, and in my own series of eight cases such a history was entirely absent in two and indefinite in a third. Experiences of this sort made me pay particular attention to this matter, and I soon found that many patients with distended gall-bladder from obstruction of its neck or cystic duct have but few symptoms. In 1921 I operated on a female patient, 44 years of age, who gave a history that she had suffered from indigestion for two months, with an attack of discomfort and soreness over the upper abdomen four days before she came under my observation. The gall-bladder was distended and reached nearly to the umbilicus, yet the patient stated that 'she could hardly say there had ever been pain.' On opening the

abdomen I found an inflamed gall-bladder full of infected mucus, the result of the impaction of a large stone in its neck. More than once I have seen similar cases where the patient would not submit to operation. The swollen gall-bladder has subsided, sometimes suddenly, and all symptoms have ceased.

" In most of such cases the gall-bladder leaks into a hollow viscus, and the stone or stones may be gradually forced into the bowel by a slow process of ulceration, as shown in the illustration (*Fig.* 11). As soon as the tension in the gall-bladder is relieved, the symptoms disappear, and the process of the gradual extrusion of the stone into the bowel is unattended with symptoms. When the gall-bladder and neighbouring parts are examined in these cases, the viscus is found to be small, thick-walled and shrivelled, and the cystic duct commonly obliterated. The opening which first forms is not large enough to allow a stone of such size as would cause obstruction to enter the intestine, but after

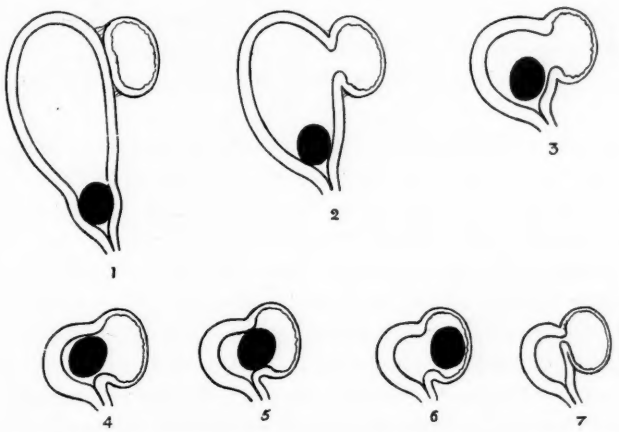

FIG. 11.—A series of diagrams showing how gall-stones may find their way into the hollow viscera. 1, A calculus is impacted in the neck of an inflamed and distended gall-bladder which has become adherent to the neighbouring bowel. 2, The gall-bladder has ulcerated into the intestine. 3, The gall-bladder drains freely into the bowel, and tension being relieved it commences to contract. 4, The process of contraction which is partly due to the development of scar tissue continues and the calculus is pressed against the opening in the bowel. 5 and 6, Show the transit of the calculus into the bowel. 7, The calculus has travelled on down the intestine, the fistula contracts, and the gall-bladder continues to shrivel.

a time the gall-bladder contracts on its contents and causes the stone to be pressed against the sinus, which is gradually enlarged by ulceration until it is big enough to let the calculus through. This is very well shown in a specimen in the College of Medicine Museum in Newcastle. [An illustration of this specimen was shown in the original paper.] The legend was as follows: ' The first part of the duodenum is laid open showing a large communication with the gall-bladder, which is occupied by a faceted calculus. Lying free in the bowel is the sister calculus, which became impacted in the ileum and caused the death of the patient from acute obstruction. The facets on the calculi exactly correspond.' In this specimen the two stones had evidently lain together with the broad facets in apposition, and the fistula is just of the exact size of the stone in the intestine. For some little time after the passage of the stone the fistula probably only slowly contracts, because in one or two

instances it has been found possible to pass the stone from the intestine back into the gall-bladder. In the specimens that I have had the opportunity of dissecting, although the stones were large ($1\frac{7}{8}$ by 1 in. and $2\frac{1}{4}$ by 1 in. respectively), the opening into the gall-bladder would only admit a forefinger and the margins were very rigid. The opening will still further contract after the stone has passed, though a communication, however small, probably always persists, forming the fistula bi-mucosa. Many experiences have confirmed this view of what happens, and all the stages of this process may be observed in the course of operations for gall-stone. *Figs.* 9 and 9A [in original paper] represent the contents of gall-bladders which I have removed. Any of these stones might have been the cause of intestinal obstruction had they found their way into the bowel. The calculus shown in *Fig.* 10 [in original paper] was removed from a suppurating gall-bladder in the case of a female patient 38 years of age. The inflamed viscus was very adherent to both the stomach and colon, and had there been no surgical intervention, it is highly probable that the large stone, which is just of the size to produce an intestinal obstruction, would have found its way by ulceration into the gastro-intestinal canal.

"Frequently when operating for gall-stones I have found communications between the viscus and some part of the bowel, and have elsewhere drawn attention to the way that these tend to be drawn out and become infundibuliform, then presenting considerable danger, for they may be inadvertently treated as simple adhesions. In July, 1909, I operated on a female patient, age 32, for the removal of a large ovarian cyst. A mass felt in the gall-bladder region was investigated, and a large stone found lying between the remains of the gall-bladder, the hepatic flexure of the colon, and the stomach, both of which had been opened by this process of slow ulceration. The calculus was removed and the patient made a good recovery. I made every endeavour to obtain a history of an attack of abdominal pain or previous abdominal illness, but she assured me that there had been none.

"This tendency of foreign bodies to find their way into the hollow viscera seems very general in pathology, and is certainly not limited to gall-stones. Some years ago I published (*The Lancet*, May 6th, 1916) some cases of 'Foreign Bodies in the Bladder resulting from Gunshot Wounds' and gave examples of foreign bodies which had slowly and gradually ulcerated into that viscus. Recently I have had a further remarkable example in the person of an ex-soldier who was wounded in May, 1917, portions of a shell being embedded in the left buttock and thigh. Two operations were performed for removal of fragments, and later for secondary hæmorrhage. He eventually made a good recovery and was discharged from the Army in July, 1918. He soon regained his usual good health, and was able to resume employment in a bank. There was never the slightest suggestion of bladder trouble until April, 1925, when he began to have frequent painful micturition. These symptoms continued until July, when he was admitted under my care. Cystoscopic examination disclosed the piece of shell, which was removed (it measured $1\frac{1}{2}$ by $\frac{3}{4}$ in.). There was only a slight cystitis, and no scar to indicate where the fragment had entered the bladder. There can be little doubt that it lodged in the soft parts outside the bladder at the time of the casualty, and

that by a slow process of ulceration it was extruded into the viscus, probably about April, 1925. Between then and the time of its removal the track by which it entered the bladder had closed, for in the process employed by nature ulceration and repair go hand in hand."

In the present case the site of impaction is unusual, for as a rule calculi causing obstruction are arrested in the small bowel. I have now operated on ten occasions for gall-stone ileus, and only in two was the impaction in the large bowel. Calculi of sufficient size to block the large bowel must be very unusual, and any stone that has passed the ileocæcal valve is probably assured of an unimpeded passage through the large bowel and to the world beyond. But I know of a case in which such a calculus stuck at the anus and had to be manually extracted. Even stones which reach the lumen of the large bowel direct must be of large size to be arrested, though, as in the small intestine, spasm may play a part, and I believe this is especially likely to occur in the sigmoid. This case adds another recovery to my admittedly fortunate series, making 8 recoveries out of 10 operations for this very serious condition.

REFERENCE.

[1] GREY TURNER, G., *Post-Graduate Med. Jour.*, 1927, Feb.–March.

THE EARLIEST SURGICAL TREATISE.*

By WARREN R. DAWSON, F.R.S.E.

Ancient Egypt is the starting-point in all studies in the history of medicine. From the literature of that venerable civilization that flourished on the banks of the Nile the earliest known medical documents have come. All that has hitherto been written of the history of medicine in ancient Egypt is mainly based upon a single document—the well-known Ebers Papyrus. This venerable manuscript was found in a tomb in 1862 together with another medical papyrus (with which this paper is concerned), a book of mathematical tables and problems (now well known as the Rhind Mathematic Papyrus), and some other documents. The two medical papyri passed into the possession of a certain Edwin Smith, an American resident in Luxor who dealt in antiquities but at the same time took an intelligent interest in them and made considerable attempts to learn the language in which the texts and inscriptions were written. The longer of his two medical papyri he sold, a few years after its acquisition, to a German Egyptologist, Georg Ebers. The delighted purchaser named his newly-gotten prize after himself, although what has become generally known as the Ebers Papyrus is always referred to in Egyptological literature prior to his purchase as 'Papyrus Smith'. Ebers published a facsimile of his medical manuscript with an introduction and glossary, but he wisely did not attempt a translation. I have elsewhere given an account of this and of the other known Egyptian medical manuscripts† : it is therefore needless to say anything further of them now beyond quoting a single paragraph from my former account :—

> The Ebers Papyrus is not a book in the proper sense of the word ; it is a miscellaneous collection of extracts and jottings collected from at least forty different sources. To call it, as Ebers did in his publication, a Hermetic Book, is entirely to mistake its nature and purpose. It consists mainly of a large collection of prescriptions for a number of named ailments, specifying the names of the drugs, the quantities of each and the method of administration. A few sections deal with diagnosis and symptoms, another passage is physiological in character and describes the action of the heart and its vessels, and the concluding portion is surgical, being concerned with the treatment of wounds and suppurating sores. Freely interspersed amongst these elements are spells and incantations.

Egyptologists familiar with the intricacies of the language have long recognized that the corruption of the text, together with its intrinsic linguistic difficulties, is such as to make an intelligible English translation of the Ebers Papyrus wellnigh impossible. This justified caution, however, has not

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* A paper read before the Osler Club, Nov. 13, 1931.

† *Magician and Leech*, 1929, pp. 73 et seq. London.

deterred others, knowing little or nothing of the hieratic script and of the language imparted by it, from rushing in where Egyptological angels feared to tread, and the result has been the production of several so-called 'translations' that are composed mainly of guesswork, and in the violence they do to grammar and syntax are mere travesties of the originals they profess to interpret. Unhappily it is on these data (and on one 'translation' in particular, which unfortunately has recently been given a new lease of life) that the medical historians have mainly drawn, with the result that an erroneous notion of the real nature of Egyptian medicine has been imparted to former generations of students, which the present generation is making some attempt to correct. The Ebers Papyrus and its kindred documents—the medico-magical papyri of London, Berlin, Leiden, California, and elsewhere—may now be dismissed from our attention, as they are merely miscellaneous collections of recipes and incantations, and (save for a few passages) they have no right to be considered as scientific medical books.

The other of the two medical papyri which belonged to Edwin Smith remained in his possession all his life, but it was not published.* After his death in 1906 the manuscript passed into the hands of the New York Historical Society. A few years ago this body invited Professor James Henry Breasted to study and edit their papyrus, and a preliminary examination so impressed Professor Breasted with the interest and importance of the text that he undertook to publish it *in extenso*. He has recently published the result of his labours in two large volumes: one containing a photographic reproduction of the hieratic text and a transcript into hieroglyphs, the other an introduction, translation, and commentary.†

It is most fortunate, not only for Egyptology but for the history of medicine, science, and human thought in general, that this important document has fallen into the hands of so competent a translator. Professor Breasted's translation may be relied on, in the main, as thoroughly accurate and sound, although naturally there are some points upon which opinions may differ. In addition to the literal translation with its elaborate philological commentary, Professor Breasted has provided for medical historians and others unacquainted with the ancient Egyptian language, a running translation of the whole text, thereby imparting to readers a general idea of the purport of the document without involving them in philological and other technical discussions.

The Edwin Smith Papyrus contains two entirely distinct groups of writings. The first is a surgical handbook, and the second is a batch of miscellaneous recipes and charms similar in nature and content to those

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* In 1864 Edwin Smith had some intention of publishing the papyrus, but the project never materialized. (Brit. Mus. Add. MSS., 31,285, fo. 130.)

† *The Edwin Smith Surgical Papyrus.* Vol. I, Hieroglyphic Transliteration, Translation and Commentary, pp. xxiv + 596, 8 plates, 4to. Vol. II, Facsimile Plates (22 collotype, and 22 line plates), folio. University of Chicago Press, 1930.

Professor Breasted had previously published three preliminary accounts of the papyrus: (1) *New York Historical Society, Quarterly Bulletins*, 1922, April, vi, pp. 4–31. (2) *Recueil d'Etudes égyptologiques dediées à la Mémoire de Jean-François Champollion*, 1922, pp. 385–429. Paris. (3) *Bulletin of the Society of Medical History of Chicago*, 1923, Jan., iii, pp. 58–78.

of the Ebers and other papyri. The latter are written on the back of the roll, the face of which is occupied by the surgical book. The main (surgical) text differs wholly from those of the previously known medical papyri (except a passage in Ebers which is similar in nature and arrangement), in that it is a continuous one and as such it is definitely entitled to be called a book; it is indeed by far the earliest known surgical treatise, and it deals systematically with the examination and treatment of wounds and fractures. Unfortunately the text is incomplete, and it breaks off abruptly in the middle of a sentence. The copyist apparently tired of his work, or perhaps he had borrowed the original and was obliged to return it to its owner before he had had time to complete his transcript. Having a large extent of blank papyrus left on his hands, he filled the free space with matter of a very different kind.* As it stands, the text comprises the details of forty-eight cases of wounds and fractures, classified in the 'head-to-foot' sequence that is common in Greek and later medical books. The injuries described begin with the top of the head and, proceeding in orderly succession downwards, reach as far as the thorax. At this point the text breaks off, but it is fair to presume that the original book, of which the Edwin Smith Papyrus is but a partial copy, completed the exploration of the body down to the feet.

The cases are presented in a systematic fashion, and each contains the following elements: (1) Title; (2) Examination; (3) Diagnosis; (4) Verdict (one of three alternatives); and (5) Treatment. A valuable feature of this text is the fact that in many cases glosses or explanatory notes have been interpolated, and these provide us with much valuable information and make intelligible to us expressions that would otherwise have remained obscure and meaningless idioms. The cases are all concerned with wounds, fractures, dislocations, and similar injuries, and the treatment is, generally speaking, appropriate and rational throughout, and contrasts markedly with the strange and often grotesque remedies familiar in the Ebers and other papyri. It must be remembered, however, that the Edwin Smith Papyrus deals with injuries of human, and therefore of intelligible, origin, and not with sickness and disease, of which the causes were entirely unknown to the ancients, and, being regarded by them as of supernatural origin, were treated by magical rather than by rational means.

The general coherence and intelligibility of the text of the Edwin Smith Papyrus has made its translation a far easier problem than is the case with regard to the other medical papyri. But the text is by no means free from obscurities and difficulties, and it abounds, moreover, in corruptions. To make its general arrangement clear, I cannot do better than select two specimens out of the forty-eight cases detailed in the document, and in making the choice I have selected two which are short in extent and relatively free from difficulties. Most of the cases are much more detailed, but are too long for convenient quotation. Case 21 is a good one for our

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* Papyrus was expensive material and was never wasted. It is consequently usual to find the space not occupied by the main text subsequently employed for other purposes. In this way it is common to find in a papyrus mainly devoted to a religious or literary text, scraps of accounts, journals, and other memoranda covering any free space that may remain.

purpose, and I here transcribe Professor Breasted's rendering of it (op. cit., p. 287) :—

Instructions concerning a split in his* temple. If thou examinest a man having a split in his temple, shouldst thou find a swelling protruding on the outside of that split, while he discharges blood from his nostril and from his one ear having that split, (and) it is painful when he hears speech, because of it :

Thou shouldst say concerning him : " One having a split in his temple, while he discharges blood from his nostril and his ear having that injury. An ailment with which I will contend."

Thou shouldst put him at his mooring stakes, until thou knowest he has reached a decisive point.

The five elements already referred to will easily be recognized. The first is the title, which always begins " Instructions for ", etc. ; the second is the examination, beginning " If thou examinest " ; the third is the diagnosis, beginning " thou shouldst say concerning him " ; the fourth is the verdict, " an ailment with which I will contend " ; and the fifth and last is the treatment. In this case the treatment consists of the remarkable expression " thou shouldst put him at his mooring stakes ". This is an idiom which occurs in several of the cases, and it would be wholly unintelligible to us were it not for the explanatory gloss which is given in Case 3, where the phrase first occurs. The gloss explaining the phrase " put (or moor) him at his mooring-stakes" is thus translated by Professor Breasted (p. 139) : " As for 'moor (him) at his mooring-stakes', it means putting him on his customary diet without administering to him a prescription." I cannot, however, help thinking that Professor Breasted has misunderstood the significance of the phrase. Surely to moor a man to a stake is a metaphorical way of expressing the idea of confining or restricting him, and although the word (*metr*) which Professor Breasted translates 'customary' may have that significance in certain contexts, its usual meaning rather implies the notion of limitation by rule, restricting. *To put a patient on his customary diet without prescribing any medicine is surely tantamount to giving no treatment at all.* The implication, therefore, of the idiom is that in cases that are to be treated by thus 'mooring' the patient, mere external applications† are of no use, but the patient's health and strength must be restored not by his free and customary diet, but by a *special* diet. In such cases, external applications must be used until sufficient improvement by the first method has been accomplished. That, at least, is how I understand the text.

After this excursus, another specimen case may be quoted. Case 22 deals with a compound comminuted fracture of the temporal bone, and it reads as follows (Breasted's translation, p. 290) :—

Instructions concerning a smash in his temple. If thou examinest a man having a smash in his temple, thou shouldst place thy thumb upon his chin (and) thy finger

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*** It may here be noted that in all Egyptian medical texts, the organs are never spoken of in the abstract as *the* heart, *the* stomach, etc., but as *his* heart, *his* stomach, etc. There is always a hypothetical patient whose case is being discussed.**

† In the Edwin Smith Papyrus ' prescriptions ' are always external applications, not doses.

upon the end of his ramus, so that the blood will flow from his two nostrils (and) from the interior of his ear having that smash. Cleanse it for him with a swab of linen until thou seest its fragments (of bone) in the interior of his ear. If thou callest to him (and) he is speechless (and) cannot speak,

Thou shouldst say concerning him: "One having a smash in his temple; he discharges blood from his two nostrils and from his ear; he is speechless; (and) he suffers with stiffness in his neck. An ailment not to be treated."

As for: "The end of his ramus", it means the end of his mandible. The ramus (*'m'.t*), the end of it is in his temple just as the claw of an *'am'e*-bird grasps an object.

As for: "Thou seest its fragments (*wš.t*) in the interior of his ear", it means that some of the fragments (*wš.t*) of the bone came away to adhere to the swab which was introduced to cleanse the interior of his ear.

In this case the third form of verdict is used, "an ailment not to be treated"—that is to say, the physician recognizes that the injury is of so serious a nature as to be beyond his powers, and that recovery cannot be expected. Of the second and third verdicts, the two cases quoted provide examples. The first form, "an ailment which I will treat" is used in simple cases where the practitioner is confident of a cure; the second implies a case that must be seriously tackled and may be successful; while the third is a frank admission that the surgeon considers the case to be hopeless. The first verdict occurs twenty-nine times, the second eight times, and the third fifteen times in the papyrus.*

The case just quoted has two explanatory glosses†: the first explains the meaning of the phrase "the end of his ramus", the forked head of which, terminating in the condyle and the coronoid process is likened to the claws of a bird. The second gloss, by its reference to splinters of bone, makes it clear that a case of comminuted fracture is envisaged.

These two cases are, as I have already said, selected solely for their brevity. Some are very long and detailed, and in the elaboration of symptoms and diagnosis they reveal a spirit of real scientific observation and inquiry. Such, for instance, are the observations on the brain. As an organ, the Egyptians seem to have attached little or no importance to it: the heart was believed by them to discharge the functions that later ages recognized as belonging to the brain. In the Edwin Smith Papyrus, however, there are recorded some notes on the brain itself. It is there recognized that the cerebral hemispheres are enclosed in a membrane. This fact could not have been learned from the process of removing the brain during the manipulations of mummification, because the custom of taking away the brain was not introduced into the technique of embalming until the Eighteenth Dynasty, long after the composition of the Edwin Smith Papyrus or its prototype. In mummies of earlier periods the brain was invariably left *in situ* and no attempt was made to remove it. Nor could the embalmers, who in the course

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* The total number of verdicts is thus fifty-three in forty-eight cases. This is accounted for by the fact that two verdicts are given (in alternative circumstances) in Cases 7, 8, 34, 37, and 47, and none in Case 9. In Case 34, the second verdict should obviously be Verdict 3, and not Verdict 1 as erroneously stated in the manuscript.

† Actually there are three glosses in this case, but for the sake of brevity I have omitted the third.

of their craft dragged out the brain piecemeal through a forced passage hewn in the ethmoid or sphenoid processes, be aware of its appearance as a whole. The remark, therefore, in the Edwin Smith Papyrus, that the brain is characterized by a series of convolutions " like those corrugations that form on molten copper ", must have been based upon the actual observation of the organ exposed by an extensive gash in the skull. A reference is also made to a wound that lacerates the " membrane enveloping his brain " so that it " breaks open his fluid in the interior of his head "—an allusion to the meningeal membranes and the cerebrospinal fluid.

Furthermore, many interesting observations are made as to the effects of brain injury : the loss of control over various parts of the body, the tension of the facial muscles, giddiness, and other manifestations. Professor Breasted, however, seems to me to be making too generous a claim for the medical knowledge of the Egyptians when he understands that their physicians recognized these symptoms as proceeding from injury to the *brain*. It seems to me that they are implicitly ascribed to injuries to the *skull*, without the definite knowledge that the brain itself is the ultimate cause of the observed effects. In some cases, indeed, the ancient surgeon seems to have been at fault : there is evidence, for instance, that it was observed in the case of an injury to the skull involving the brain, that the eye is affected on the same side as the injury to the skull, and partial paralysis of the leg resulting from a head injury is also noted as being on the same side as the wound in the skull that caused it. There are two possibilities open here : either that the text is corrupt, and instead of reading 'on the same side', the words 'on the opposite side' were originally written ; or, alternatively, the observer may have had before him a case of *contre-coup*, from which he wrongly generalized. Be this as it may, it is most interesting to find, in a book written more than thirty-five centuries ago, even the bare observation (be it rightly or wrongly stated), that injury to the skull (brain) produces manifestations in areas far removed from the seat of the wound, and that the side of the body in which such manifestations occur should be noted in relation to the side of the head that is injured.

Another observation of great interest is that recorded in Case 31, where it is noted that injury to the spinal column (in this case a cervical dislocation) may cause a prolonged priapism, sometimes followed by *emissio seminis*. In the description of the symptoms of the same case, it is also said that " his flesh has received wind ". This is evidently a reference to meteorism, when, owing to intestinal paresis, undischarged gases cause inflation of the abdomen.

Throughout the papyrus, the details of such cases are sufficiently fully stated, or explained in the glosses, to enable us to recognize them and to name them in terms of modern anatomy, physiology, or pathology with considerable precision.

Some of the cases are of simple flesh wounds that do not involve the bones or indeed any tissues other than the superficial. Amongst such cases are wounds in the scalp, chin, brow, nose, external ear, and lips. Injuries that are more deeply seated include a wound in the throat that penetrates the gullet, a gaping wound in the shoulder associated with inflammation and

fever, and gaping wounds in the head and neck penetrating the bones of the skull and cervical vertebræ respectively. The bone injuries mentioned include: *perforation* of the skull and its sutures, of the maxillary and zygomatic processes, and of the temporal bones; *fractures* (simple, compound, or comminuted) of the skull, of the nasal cartilage, of the maxillary, zygomatic and mandibular bones, of the humerus, of the clavicles, of the ribs, and of the cervical vertebræ; and *dislocations* (and sprains) of the mandible, of the cervical vertebræ, of the clavicles, and of the sternocostal articulations. These, together with localized tumours and abscesses, make up the forty-eight cases of the Edwin Smith Papyrus.*

A few words may now be said as to the methods of treatment employed. The retracted edges of gaping wounds, if small, were drawn and held together by means of adhesive plaster, or, if large, by stitching. Minor wounds were treated by simple bandaging. Raw meat was often used (as it is to-day) as an external application to reduce swelling or inflammation, but it is specifically stated that it is to be used only on the first day. An ointment, the basis of which was honey, was freely used, often applied on swabs. For bone injuries, reduction of dislocated or fractured bones was frequently resorted to, with or without the subsequent use of splints. The rigid splints (of which actual specimens have been found) were tubular cases of palm-fibre,† but in some cases supports, in the form of stiff linen rolls, were used. The actual surgical treatment was often accompanied by special diet, or the treatment in some cases was by diet in preference to therapy, a point that has already been alluded to in this paper. The ointments generally consisted of honey mixed with pounded herbs or with decoctions of herbs. The latter are often quite valueless by modern standards, although amongst them may be recognized a few that have well-marked astringent or antiseptic properties. The choice of the herbs and other drugs was still largely influenced by magic.

Reference has already been made to the glosses in the Edwin Smith Papyrus. They are of great interest and were evidently intended to explain the significance of expressions or terms that were considered to be too technical to be understood without such aid or that had become obsolete since the book was first composed. Twenty-nine out of the forty-eight cases have one or more of these explanatory glosses, of which there are altogether sixty-nine. It may be noted in passing that certain of the passages in the Ebers Papyrus that contain anatomical or physiological particulars are accompanied by glosses; but that long document (physically nearly seven times as long as the Edwin Smith Papyrus) contains altogether only twenty-six glosses, many of which have been displaced by ignorant copyists and thereby rendered scarcely intelligible. The glosses in the Edwin Smith Papyrus show that their composer was actuated by a really scientific interest in the cases before him. He often goes into details regarding the hopeless cases that he could not remedy, which shows that his interest did not cease merely with the

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*There are twelve cases of flesh wounds, and thirty-three of injuries to bones and articulations.

† G. Elliot Smith "The Most Ancient Splints", *Brit. Med. Jour.*, 1908, March 28, p. 732.

practical side of treatment, but that his curiosity was stimulated to investigate what he could not cure. This fact indicates an approach towards the Hippocratic method. In his editorial introduction to the Hippocratic *Epidemics*, Mr. W. H. S. Jones says* :—

> Of the forty-two cases, twenty-five end in death, very nearly 60 per cent. The writer's aim is not to show how to cure—treatment is very rarely mentioned—but to discover the sequences of symptoms, to set down the successes and failures of Nature in her efforts to expel the disease. The physician is acting, not *qua* physician, but *qua* scientist ; he has laid aside the part of the healer to be for a time a spectator looking down on the arena.

Of the text itself of the Edwin Smith Papyrus, the aim is purely practical ; its function is to describe symptoms and to indicate treatment. To the original author of the book, who probably lived at the time the Pyramids were being built, the lofty motives of the Hippocratic School cannot be attributed, but the glosses show that upon some later commentator was dawning the inspiration to make original research into causes and effects. In his notes, garbled and corrupt as they often are in the form in which they have come down to us in this the earliest surviving copy of them, are to be discerned the germs of that spirit of philosophical inquiry that only the genius of the Greeks with their powers of abstract thought (a power totally lacking in the Egyptians, gifted though they were) could bring to the stage they actually attained—that of forming the foundation upon which many generations of thinkers and inquirers have erected the edifice of modern medicine.

The Edwin Smith Papyrus, containing as it does the earliest practical handbook of surgery together with the notes of an ancient forerunner of Hunter, can justly claim to be the oldest scientific book. Professor Breasted declares that it destroys the general belief, in which he formerly shared, that Egyptian medicine had its origin in magic. With this general opinion I find it impossible to agree. It does not in the least detract from the importance and interest of the Edwin Smith Papyrus to recognize that while it undoubtedly affords evidence that a serious attempt was being made to understand the elements of anatomy and physiology, yet it must be clearly borne in mind that it is a book that deals only with wounds and fractures—injuries of palpable and intelligible origin—and not with diseases, for which perforce magical methods had to be employed, because the causes of illness and disease were to the ancients invisible, impalpable, and unknown. A wound or an injury caused by a fall or other accident, or by a weapon or a tool, was well understood and treated by rational means ; but the causes of headache and fever, of skin diseases or swellings, and of countless other maladies, were wholly mysterious and attributed to supernatural agencies. Two brothers might on the same day come before the doctor at Memphis or Thebes, the one for treatment of a dagger-wound in his breast, the other for a painful and irritating rash affecting the same region of the body. The cause of the one was self-evident ; the cause of the other was a mystery, and the treatment in the two cases differed in its nature accordingly.

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* *Hippocrates*, i, p. 144 (Loeb Classical Library, 1922).

Magic never lost its hold upon medicine, and it has retained its grip even to this day to a far greater extent than is generally recognized. Whilst the beginnings of real research were lifting medicine out of the domain of the magician, the treatment of common ailments still and for long ages continued to come under his eye. The medical and the magical marched side by side, and the same age produced both the Edwin Smith and the Ebers papyri, with their widely-differing contents. Indeed, on the back of the Edwin Smith Papyrus itself there is written a collection of charms and prescriptions similar in character to those that fill page after page in the other so-called 'medical' papyri. The ancient owner of the Edwin Smith Papyrus saw no incongruity in having in one and the same note-book elements that appear to us of to-day as absolutely antagonistic in nature and content. One might imagine, as a parallel, a modern student taking simultaneously and equally seriously the utterances of Paracelsus and of Hunter. I will conclude by a brief reference to these miscellaneous jottings that fill the verso of the Edwin Smith Papyrus.

There is first a series of eight incantations against plague. These are grouped under such headings as " Incantation for exorcising the wind of the pest of the year ", or, " Another for exorcising the plague-bearing wind,* the demons of disease, the malignant spirits, messengers of Sekhmet.† " These are all charms or incantations to be recited over images of gods or other talismans, and are purely magical. The next item is a prescription for a female complaint, and as I cannot agree with Professor Breasted's translation of it, I will offer a rendering of my own :—

> If you examine a woman who suffers in her stomach, so that for her the menses do not come, (and) if you find something in the upper part of the vagina, you say concerning her : " It is a clot of blood‡ in the womb." You prepare for her balsam, $\frac{1}{16}$; grease, $\frac{1}{8}$; sweet ale, $\frac{1}{8}$; warmed and drunk for four days ; besides preparing for her (the prescription for) the discharge of blood, (namely) oil, cumin, stibium, sweet frankincense. Mix and anoint the pubes§ therewith frequently.

As I understand the passage, it is clearly the treatment of irregularity in menstruation due to clotting. The symptoms are nausea and local pain. For the relief of the former an emetic is prescribed, and for the latter an ointment to be rubbed on the lower part of the abdomen to relieve the pain in the vagina and to provoke the flow by massage.

After this, another prescription for menstruation follows, this time employing a fumigation. The concluding recipes are all pure quackery and differ little either in substance or in kind from the boldly-advertised trash of the seventeenth and eighteenth centuries in Europe, which under a thinly disguised exterior is still prevalent to-day. The titles of these recipes alone sufficiently indicate their character : " Recipe for Transforming the Skin ",

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* That plague was carried by the air remained one of the main tenets of medical opinion for many centuries later. It was the dominating principle that ordered the closing of windows and the lighting of fires in the streets in the Great Plague of 1665.

† A malevolent goddess.

‡ *Shen'w n snf*, lit., ' an obstruction of blood '.

§ Reading *Kns* for *Ks* as written in the MS. in error.

"Recipe for Beautifying the Face", and lastly, "A book for Transforming an Old Man into a Youth". The quest of personal loveliness and eternal youth is as old as the cradle of civilization.

We must not, however, allow the quackery of the verso of the Edwin Smith Papyrus to prejudice our minds or to detract from the rational and valuable surgical treatise of the recto. A careful study of the forty-eight cases that have survived must convince an unprejudiced reader that the Surgical Book of the Edwin Smith Papyrus has a just title to be known and respected as the earliest scientific book that time has bequeathed to posterity. Medical historians must revise their estimate of Egyptian medicine in the light of this new accession.

A CASE OF TRANSPLANTATION OF THE RIGHT URETER INTO THE CÆCUM AND OF THE LEFT INTO THE SIGMOID FIVE YEARS AFTERWARDS.

By C. E. L. BURMAN, Pietermaritzburg, Natal.

As far as I am aware the cæcum has not been used as a reservoir for the urine, and it would appear that there are certain advantages to be gained by such a procedure in preference to the recognized sigmoid route: (1) The chances of ascending infection, although considered not tenable to-day, are certainly less than in the sigmoid. (2) The urine passes into a more or less definite receptacle. (3) Absorption of water takes place during transit of urine along the whole length of the large bowel, with consequent less desire to pass urine. (4) There is no chance of solid contents interfering with the flow of urine from the kidney, as the contents in this area are fluid. (5) The cæcum is easily fixed in a convenient position. (6) The appendix stump orifice is a convenient and comparatively easy and safe method of anastomosis by means of the ink-well method. (7) There appears to be no irritation when the urine passes into the cæcum, as the acidity is quickly neutralized by the alkaline contents from the ileum.

The following case supporting the above statements seems to be justified:—

History.—In January, 1926, J. V., female, age 16 years, had pains in the abdomen for two days and was then taken by her people to see a European doctor, who drew off a large quantity of urine and told her she was pregnant and in labour. The patient herself was not aware that she was pregnant until told by the doctor. This may seem a difficult statement to believe, but in the raw native I have seen it more than once. The labour was apparently difficult, and craniotomy was carried out. She was on her back for two months following the confinement. I saw her on June 25, 1926, when she complained of being constantly wet and of inability to hold her urine.

On Examination.—Considerable ulceration of the posterior and lateral walls of the vagina was revealed and a large hole in the anterior wall of the bladder. By retracting the sides of the hole the ureteric orifices could be plainly seen. The whole urethra except for the external urinary meatus was entirely absent. A plastic operation was carried out and a catheter tied into the bladder. The catheter came out and was not replaced, with the result that the newly constructed urethra gave way and the patient was no better off. Examination on July 16 showed that even the small piece of urethra had been obliterated and the external urinary meatus was represented by a dimple. It was decided that the only possible solution was a transplantation of the ureters, and the following operation was carried out on the right ureter the same day.

First Operation (July 16, 1926).—An oblique muscle-cutting incision was made in the right iliac region of sufficient length to give good exposure of the cæcal and ureter area. The ureter was isolated and traced down to the entrance at the bladder and excised at the ureteric orifice and clamped at the extreme end. The hole in the bladder was closed and the ureter brought out of the wound and covered with gauze. An incision was then made in the posterior parietal peritoneum extending from the ileocæcal junction along the brim of the pelvis as far as the caput cæci. The appendix was clamped and removed in the usual way and the stump opened up. The ureter was brought through the tunnel and inserted, after freshening up the end, through the orifice of the appendix stump for about 2 in. into the cæcum. The peritoneum was invaginated all round the ureter and secured by four interrupted sutures of Pachenstecker thread catching up the peritoneum and ureter with each suture. Another row of interrupted sutures was inserted around this area so that the ureter was ink-welled into the appendix stump. The cæcum was fixed by suturing it into the incision made into the posterior parietal peritoneum. The incision for isolation of the ureter was closed with a few sutures, a drain being placed in the operation area, and the abdomen closed.

After-progress.—The drain was removed on the third day after operation. Recovery was uneventful, and the patient was discharged from hospital on Aug. 2. Nothing was heard of her until Oct. 4, 1929, when she reported for examination. Her general condition was excellent, but she still complained of being wet—the result of the left ureter. Operation was advised and accepted.

Second Operation (Oct. 8, 1929).—Indigo-carmine (2 c.c.) was injected intravenously just before the administration of the anæsthetic. The cæcum was exposed through an independent incision on the right side and punctured with a needle with a view to ascertaining whether coloured urine was present in the cæcum, but none was obtained. The left ureter excreted coloured urine. An oblique muscle-cutting incision was now made in the left iliac region and the abdomen opened. The right kidney was first palpated and found normal in size and consistency. Transplantation of the left ureter into the sigmoid was now decided on, but the ureter could not be located and the operation was abandoned. The patient went home *in statu quo* on Oct. 22.

After-progress.—The girl's whereabouts could not be traced until December, 1931, when I was able to have her admitted to Grey's Hospital, Pietermaritzburg. On Dec. 5 examination of the vagina was undertaken without an anæsthetic, but all that could be made out, owing to the tenderness and pain produced, was a sodden condition of the vulva and inner sides of the thighs, a marked contraction of the vaginal orifice and vagina, ulceration of the lateral and posterior walls, and that the urine was welling out of the bladder and forming a puddle in the vagina. The urinary meatus was represented by a small depression drawn over to the left side. On Dec. 8 radiograms of both kidneys were taken and the radiologist reported: "Gas shadowing obscures to some extent the right side (upper half). Dye appears in both pelves. The appearances suggest the presence of a double ureter on the right side (upper half). Slight diffuse opacity in the cæcal and ascending colon suggesting the free flow of uroselectan into the cæcum."

THIRD OPERATION (Dec. 10, 1931).—The old scar on the left side was excised and the abdomen re-opened. Considerable omental adhesions and matting of the sigmoid were encountered. The right kidney felt normal. The

FIG. 12.—Two minutes.

FIG. 13.—Five minutes.

FIG. 14.—Fifteen minutes.

FIG. 15.—Forty-five minutes.

ureter was again found difficult to identify, but it was finally located somewhat farther out than usual and traced upwards and downwards until a good 3 in. was isolated. The sigmoid was mobilized and the ureter transplanted by using a combination of Coffey's and Stiles's methods. No ureteral catheter was used, but a bed in the sigmoid was prepared by an incision of the peritoneal and muscular coats for about 2 in. and the ureter drawn through a small incision at the distal end and fixed into the bowel. Two further sutures were passed through the outer wall of the ureter and the peritoneum of the sigmoid, and the bed and ureter were finally buried with a continuous suture. A drain was passed down to the area and the abdomen closed.

AFTER-PROGRESS.—Uneventful until the sixth day following operation, when the patient was suddenly taken ill with an acute pain in the left axillary region with a rise of temperature to 102·6° and a pulse-rate of 120. The next day she complained of pain over the right side of the abdomen and distension. A slight watery discharge was noticed on the dressing, but a fair quantity of urine was passed by the rectum. A large poultice was applied for one hour. Relief was almost immediately obtained by the passage of flatus, fæces, and urine and diminution of abdominal distension. The watery discharge, now more suspicious of urine, was more profuse, but it quickly grew less, and by Dec. 27 the patient was dry. The daily output of urine, from now onwards, was 50 oz. Tolerance varied from one to five hours. The wound was soundly healed.

FIG. 16.—Seventy-five minutes.

Jan. 3, 1932.—Examination per vaginam was carried out by the insertion of a gauze soaked in 20 per cent cocaine solution, and it was found that the ulceration was considerably improved. The vagina was still markedly contracted, and the hole in the anterior wall of the bladder admitted quite easily the forefinger, allowing digital examination of the bladder. The failure of the original plastic operation was undoubtedly due to the inability to reconstruct the urethra.

Jan. 5.—Intravenous pyelography was carried out, and the report from the radiologist stated : " Slight excretion of the dye is noted in the left kidney 2 minutes after injection. At the 5-minutes examination slight tendency to hydronephrosis is seen in the left kidney and marked hydronephrosis in that of the right. The right ureter is not seen filled with dye in 15 minutes, but the pelves and calices are well defined. At the 45-minutes examination optimum concentration of the dye is present in the left side and marked kinking

of the ureter (dilated) is evident on the right side. The course of the ureters is seen in their entirety on both sides." (*Figs.* 12–16.)

Jan. 16, 1932.—The patient was discharged from hospital in excellent health (*Fig.* 17) and went to her own home up country. At the end of the

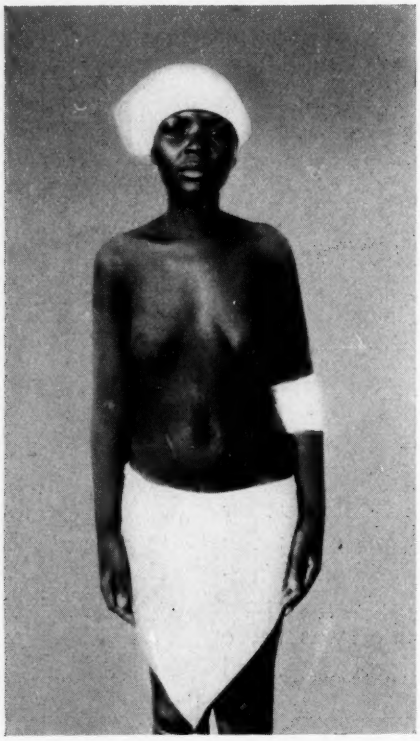

FIG. 17.—Condition of patient on discharge from hospital.

month she was suddenly taken with purging and vomiting, and from this she died on Feb. 3. There was no post-mortem.

COMMENT.

The marked dilatation of the right ureter with kinking and the associated hydronephrosis noted after pyelography following operation as compared with the pyelogram before operation suggests that the acute attack of pain complained of in the right side on the sixth day after the last operation was the cause of this unfortunate accident. The reason of this appears hard to explain as the patient had been perfectly well ever since the transplantation in 1926. It may be that the somewhat severe reaction which followed the administration of liquorice powder on the sixth day to obtain bowel evacuation caused an upheaval in both the recent and previous operation areas.

LEUCOPLAKIA IN THE URINARY TRACT, WITH A REPORT OF A CASE.

By H. P. WINSBURY-WHITE,

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Leucoplakia involving the urinary tract is uncommon, but of recent years many more cases have appeared in the literature than were formerly reported, suggesting that the disease at one time often escaped notice. Writing in 1929 Patch[1] found 123 reported cases involving the kidneys, ureters, and bladder.

CASE REPORT.

The following are the facts relating to a case which came within my personal experience :—

History.—The patient, a married man, age 41 years, with three children, was first seen by me in February, 1930, complaining of increased frequency of micturition, and discomfort at the end of passing water. In 1913 he had hæmaturia for three months. He was admitted to St. Thomas's Hospital. A note from the Surgical Registrar about his condition at this time is as follows : " In 1913 this patient had excision of a so-called papilloma of the bladder, the microscopical report of which was—chronic inflammatory. He was in again in 1921, when a cystoscopy revealed a bright red mucosa, the bladder only taking 4 oz. Lymph flakes, but no phosphatic deposits, were seen. He was classified as a case of phosphaturia owing to the heavy deposit of phosphate in his urine."

The patient said that since 1914 he had seen sediment in his urine, and had been troubled with frequency of micturition, urgency, and dysuria. In February, 1930, he could hold his water for about two hours. Up to this time he had for many years carried on his occupation of a labourer. He denied ever having had syphilis or gonorrhœa.

On Examination.—The patient was of spare build, but otherwise looked well. Both epididymes were somewhat thickened but not adherent to the testes. There was no evidence of urethral discharge. On rectal examination nothing abnormal could be noted. The urine was turbid and contained débris, which was more marked in the second than in the first glass. A detailed report on the urine showed the presence of amorphous and triple phosphate and a moderate number of pus cells and bladder débris entangled in mucus on microscopical examination, while a strong growth of *B. coli* resulted from cultivation. Investigations for tubercle bacilli were carried out on a number of occasions without revealing the presence of these organisms.

An endeavour to cystoscope the patient was unsatisfactory, as on attempting to fill the bladder the fluid at once returned outside the cystoscope, and no more than one ounce was retained. Under these conditions the only information obtained was that the vesical mucosa was pale and covered with masses of mucoid material. Posterior urethroscopy showed the roof of the posterior urethra to have the same pallor as the bladder.

A plain radiographic examination of the urinary tract showed no evidence of calculus. Further radiograms after the intravenous injection of uroselectan showed no further abnormality in outline of the kidneys or ureters than a very slight degree

of dilatation on the right side, but the poor density of the shadows indicated reduced renal function on both sides. Blood examination showed Wassermann negative, Vernes 0, and resorcin test with an optic density of 95.

OPERATION.—I decided to investigate further by opening the bladder suprapubically. On doing so, I found a thin-walled organ which had all the appearance of a normal capacity, but presenting a most remarkable appearance (*Fig.* 18). The mucous membrane was uniformly bluish-pink in colour, except on the trigone, where ulceration was present. At the margins of the incised vesical mucosa the thickening of the vesical epithelium was apparent. The ulcerated area roughly occupied the trigone, with several finger-like processes extending beyond these limits, one of them reaching towards and beyond the left ureteric orifice. There was no undermining of the epithelium bordering the ulcer. On the contrary, the edges were slightly rounded from œdema. The thickened epithelium in the vicinity of the internal urinary meatus was somewhat corrugated, doubtless from the contraction of the sphincter.

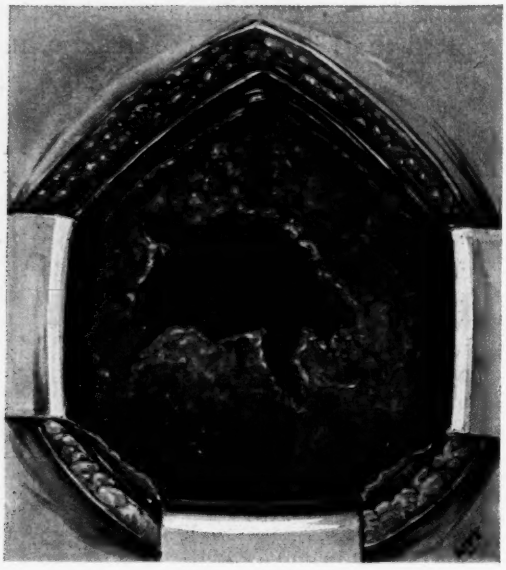

FIG. 18.—View of bladder base through a suprapubic exposure, showing internal urinary meatus, and the extensive ulcer on the trigone.

A striking feature of the mucous membrane was the way the whole surface except the trigone became and remained completely dry after swabbing. Scattered particles of non-adherent phosphatic grit were removed by the same process. The ultimate result was a dry, glistening, bluish-pink appearance, suggesting the new skin formed over a somewhat recent scar.

An attempt was made to catheterize the ureters during this investigation, but the difficult approach to the left orifice made it impossible on this side, while the opposite orifice was so obscured by thickened epithelium that it could not be found.

After removing portions of tissue for microscopical examination from the margin of the ulcer, and from the bladder wall at the site of the vesical incision, the wound was closed round a tube draining the bladder. The microscopic sections both showed that the epithelial lining of the bladder was squamous in type, with keratinization of the superficial layers, a well-marked stratum granulosum, and well-developed and numerous tongues of epithelium projecting deeply into the underlying connective tissue. In places masses of epithelium isolated from the overlying layer indicated the marked degree of epithelial hyperplasia (*Fig.* 19). The penetration of these cell masses could not be found so deeply as the muscular coat,

and their isolation from the superficial epithelium is readily explained by the manner of cutting of the section with the knife. No area could be identified which would justify the diagnosis of malignancy, but the evidence of marked epithelial activity indicated the tendency towards malignant transformation.

RADIUM TREATMENT.—Up to twelve weeks after the operation the suprapubic fistula had failed to close completely. In consultation with my colleague, Mr. Stanford Cade, it was decided to implant radium under the ulcerated area. Mr. Cade undertook this, and I had the benefit of assisting him. I was able to note that the condition of the ulcer on the trigone was not appreciably altered from that which existed at the previous cystotomy. Four 1·3-mgrm. radium needles were inserted beneath the ulcerated surface and left in position for seven days. The suprapubic fistula was soundly closed in seven weeks, and the patient was discharged from hospital and returned to his work.

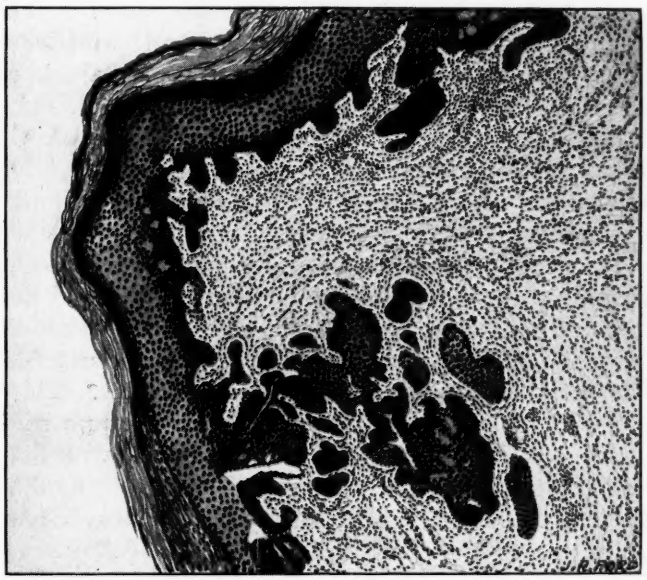

FIG. 19.—Microscopic section showing marked hyperplasia of vesical epithelium. (× 50.)

Before he left hospital cystoscopy showed the same appearance of the bladder as before, but with regard to the trigone, although some œdema was noted, no ulceration could now be seen.

SUBSEQUENT PROGRESS.—Nine months later the patient responded to an invitation to report for a further examination. After discharge from hospital he had resumed his manual work, and was still in employment. Although he was a little thin, he had the appearance of fairly good health. On this occasion it was found that the patient's symptoms were practically the same as before I first opened the bladder. It was noted, however, that there was a slight mucopurulent urethral discharge. This apparently had been present for several months. No gonococci were to be found in subsequent bacteriological examination.

In accounting for the discharge one remembered that the leucoplakia had been observed in the posterior urethra on urethroscopy. The urine was still turbid and contained débris. An attempt at cystoscopy was no more successful than on previous occasions, the irrigation fluid being ejected at once outside of the cystoscope.

In this case the outstanding feature was the uniform distribution of the change over the whole of the vesical mucosa except for the trigone, which

was ulcerated. Microscopically there was a conversion of transitional epithelium into the squamous type.

Usually all stages between the normal and well-developed squamous epithelium can be found if sections are made in different localities. The surface has a layer of varying thickness of cornified epithelium. Instead of a uniform stratum of about four layers of stratified epithelium which normally lines the epithelial surface, there is a considerable depth of squamous cells with down-growing processes extending well into the underlying connective tissue in places. Microscopic sections taken through a portion of tissue where carcinoma has developed indicate the malignant condition as but a further stage of the epithelial change. Therefore the precancerous nature of the leucoplakia seems established. Patch found thirteen cases in the literature in which squamous-celled carcinoma and leucoplakia were found simultaneously in the kidneys, ureters, or bladder. With regard to the bladder in particular, a number of cases have been reported in which the leucoplakia commenced in, and in some cases was confined to, a vesical diverticulum.

The most acceptable theory of the etiology of leucoplakia is that it is a deficiency disease. Two investigators, Wolbach and Howe,[2] found extensive epithelial metaplasia in vitamin A deficiency, which was not confined to, but included, the urinary tract. The changes noted had the features of leucoplakia. More recent experimental work by McCarrison[3] seems to confirm this view. Working with, not only large numbers of rats, but also guinea-pigs, rabbits, pigeons, and monkeys, he has been able to demonstrate the fact that diets with vitamin A deficiency produce amongst other diseases many instances of keratinization of the epithelium of mucous surfaces. This included the bladder, ureters, and renal pelves. The diets he used were composed mainly of cereal grains or cereal products and vegetable fats, with little or no milk, butter, and fresh vegetables. Of all the faulty diets he has used, that composed of white bread, margarine, tea, sugar, jam, preserved meat, and scanty overcooked vegetables proved the most injurious. The significance of this fact is that such a diet is common in poor communities.

Wilson and Du Bois[4] in a post-mortem on a child that died of vitamin A deficiency found widespread keratinization of epithelium in glandular tissue and of mucous surfaces, including the renal pelvis. This is a valuable piece of evidence linking together experimental and clinical findings. Previously two views attempting to explain the etiology commanded attention: one, that the abnormal epithelialization is a result of misplaced embryonal rests of ectoderm; the other, the theory of metaplasia which attributes the cornification to irritation. The latter possibility seemed to find support from the fact that so many cases were associated with chronic infection.

The obvious difficulty in determining the true relationship between infection and epithelial changes is to know which existed first. If infection is to be considered the cause rather than the effect, the question naturally arises why leucoplakia is so comparatively rare while infection is common. Examination of the bacteriological reports dealing with the infections has thrown no light on the etiology, as a great variety of organisms have been described. But McCarrison has shown that infection is also a common result of deficiency in diet. This evidence therefore considerably weakens the theory

of metaplasia. This investigator maintains that the epithelial changes noted are independent of infection, though this is often superimposed on them, and that they are in the nature of breaches in the body defences against bacterial invasion. He regards vitamin A as an anti-infective agent, and vitamins B, C, and D as having the same qualities, which they exercise in a different way.

Histological examinations of renal pelves not the seat of leucoplakia have revealed many instances in which the tendency towards the condition has been observed, but without giving any clue to the cause. Lavonius[5] microscoped 150 renal pelves and demonstrated 5 cases in which leucoplakia might originate. Leber[6] observed widespread evidence of epithelial proliferation in a girl of four months. The patient had xanthoma of both conjunctivæ, and hyperplasia of the epithelium of the renal pelves. Lubarsch[7] in 160 autopsies found 6 cases with islands of leucoplakia, while Heyman[8] noted small circumscribed areas of cornification in 10 of 20 cases.

The epithelial lining of any part of the urinary tract may be the seat of leucoplakia. With regard to the kidneys and ureters, the disease is not uncommonly bilateral. In hydronephrosis small areas of cornification are sometimes seen. More cases have been reported involving the bladder than the renal pelvis. Hennessey's analysis of 80 cases gave 42 for the former and 24 for the latter. In the remaining cases the lesions were scattered about the urinary tract.

The naked-eye characters of the lesions are most frequently displayed as single or multiple raised grey patches of varying sizes, or there is a uniform hyaline appearance of the mucous membrane of the whole or a portion of the part involved.

With regard to age, Hennessey[9] found in reviewing 74 cases that this varied between 4 months and 71 years, with an average of 38 years. The same writer found an incidence of three males to one female.

The length of time covered by the symptoms varies enormously in reported cases. Kutzmann,[10] in reviewing 67 cases, found it ranged between 3 weeks and 35 years. The symptoms in any given case will be influenced largely by the seat of the disease and the nature of the conditions which accompany it, such as stone, tuberculosis, vesical diverticulum, hydronephrosis, and the degree of infection. No symptoms, therefore, can be put forward as being characteristic of leucoplakia. However, relating to both bladder and kidneys, signs of chronic infection are the commonest features which have been noted. Hæmaturia is frequently noted. Kutzmann reported this symptom in 35 per cent of 67 cases he analysed. In my own case, in which as far as one knows the disease is confined to the bladder and posterior urethra, hæmaturia was one of the first complaints, otherwise the symptoms were continued frequency of micturition, terminal discomfort on micturition, turbidity of the urine, and latterly a slight mucopurulent urethral discharge.

When the disease is confined to the upper urinary tract the true diagnosis will only be made by an inspection of the parts such as is possible with exploration of the kidney, nephrectomy, or at autopsy.

When there is leucoplakia of the bladder, cystoscopy may prove unsatisfactory from the fact that it may be impossible to get the bladder to retain sufficient fluid for this examination to be carried out. In these circumstances

it may be impossible to locate the ureteric orifices. This was the state of affairs in my case, and in several other cases that were reported.

When a view of an area of vesical leucoplakia is obtained, the outstanding features are the absence of visible blood-vessels on the mucosa, which is pale or greyish in aspect. In other cases actual raised pale plaques and areas of ulceration have been seen.

When the upper urinary tract is the seat of leucoplakia, provided that unilateral renal disease has been established, nephrectomy is generally undertaken for the accompanying condition, which usually dominates the clinical picture. If the disease is bilateral, then it is impossible to eradicate it. For the lower urinary tract no curative treatment is known.

Claims have been made by one writer of the benefit of antisyphilitic treatment. These have not been endorsed by others. Radium and electrocoagulation have been helpful as palliative measures.

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PREVASCULAR FEMORAL HERNIA.

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Several unusual forms of femoral hernia, differing from the ordinary one in some definite anatomical particular, have been described. These are: (1) External, or Hesselbach's, hernia, situated external to the deep epigastric artery and associated with an inguinal hernia; (2) Hernia through Gimbernat's ligament, or Laugier's hernia; (3) Pectineal, or Cloquet's, hernia; (4) Retrovascular hernia; (5) Prevascular hernia. All these are rare, and most of them are very rare, but one, the *prevascular hernia*, is probably less uncommon than is usually supposed. This view was expressed in 1924 by L. F. Watson,[1] of Chicago, and gained support from the previous writings of Moschcowitz[2] and Cevario.[3] A number of instances have been reported in the literature, but none, as far as I know, in this country, so that a brief account of my experience may prove of interest.

The usual theory of the origin of femoral hernia attributes it to the presence of a congenital peritoneal diverticulum on the inner side of the femoral vein. This diverticulum may be supposed to be formed by being carried out with the vessels as they grow down into the limb bud of the embryo. Usually the diverticulum is shallow, but is increased by intra-abdominal pressure so that it grows in the line of least resistance—that is, down the femoral canal and into the thigh through the saphenous opening. This is the state of affairs in the vast majority of femoral hernias, the fundus of the sac being in Scarpa's triangle, and the neck in the femoral canal. Now if this theory of the common origin of femoral hernia is correct, it might be supposed that very occasionally a much longer peritoneal diverticulum might be dragged out with the growing vessels, and, accompanying them down the thigh, be found within the femoral sheath in front of the vessels. The fundus of the hernial sac would then be far below the level of the saphenous opening, and would never tend to emerge through it. This would in fact constitute the condition of prevascular femoral hernia, an example of which has come under my observation as follows:—

Case 1.—A young labourer, age 23, attended at the City of London Truss Society on Nov. 25, 1924. He complained of a lump in the right groin, which had given him occasional pain and one attack of severe pain and retching. On examination he was found to have a right femoral hernia, containing loculated fluid in the fundus, so that the hernia was clinically 'irreducible'. He had never noticed anything abnormal on the left side, but in the course of my routine examination I detected signs of a reducible hernia in the centre of Scarpa's triangle immediately in the line of the femoral vessels. Nothing abnormal was to be seen, but an impulse could be felt when he coughed, and reduction of the very slight swelling then formed was accompanied by the sensation usually produced by the slipping back of an epiplocele.

An operation was performed on Dec. 5, and on the right side a loculated femoral hernia was exposed in the usual position, the neck lying in the saphenous opening and femoral canal. On the left side no hernial sac could be found in this position, but the femoral sheath appeared wider than usual. A vertical incision was therefore made in the centre of the sheath, and a long narrow peritoneal sac was exposed as it lay on the vessels inside the sheath. The fundus was dragged up through the incision in the sheath and the appearance was then as shown in *Fig.* 20. The sac was 6 cm. in length, so that it extended some way below the level of the saphenous opening, and was 2 cm. wide at its origin. It was found to be empty when opened, and it was removed by the inguinal route in the ordinary way. The patient was

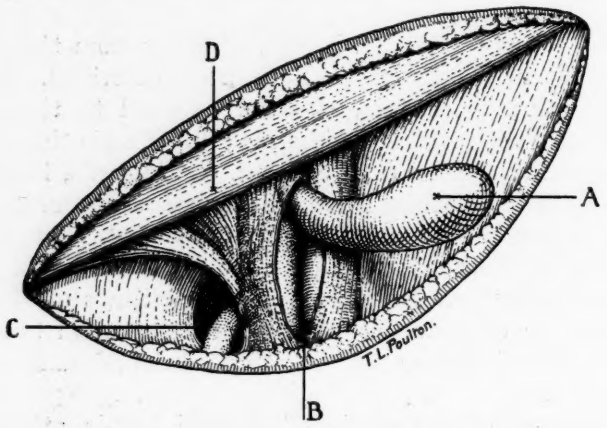

FIG. 20.—*Case* 1. Dissection of congenital prevascular femoral hernia. A, Fundus of sac; B, Femoral sheath; C, Saphenous opening; D, Inguinal ligament.

young and muscular, so that the manœuvre of approximating the lower edge of the internal oblique fascia or the conjoint tendon to the pectineal fascia would have produced great tension in these structures. A plastic operation, such as I have described elsewhere,[4] was therefore carried out on both sides, a flap of the internal oblique layer of the anterior rectus sheath being stitched to the ileo-pectineal line. On the left side the vessels were retracted outwards further than usual, and four sutures were used, so that the vessels when released came into very close contact with the outer edge of the flap, which was also sutured to the inguinal ligament in front of the vessels. The patient made a normal recovery, and has been doing heavy work ever since without any sign of recurrence on either side (7 years).

Moschcowitz claims to have described the first case "operated upon and studied *in vivo*" in 1912, and the above description agrees with the condition found by him. It is noticeable that in this instance the congenital origin of the hernia seems to be unquestionable, since the fundus, as shown in the drawing, is appreciably wider than the neck, which would scarcely be the case had the hernia been acquired. It offers therefore a striking contrast to another instance of prevascular femoral hernia which I believe had been acquired.

Case 2.—A man, age 60, was seen at the City of London Truss Society in March, 1931. He complained of a hernia on the right side, which again was in Scarpa's triangle immediately over the femoral vessels. In this patient, however, the hernia was conspicuous when he was standing, and formed a roughly triangular pouch with its base upwards, as shown in *Fig.* 21. His tissues exhibited a well-marked degree of senile laxity, and the hernial sac appeared to have formed in the upper part of

the femoral sheath as the connective tissues sagged, the 'neck', or opening, being much wider than the fundus. A lesser degree of the same condition was noticed on the left side. The patient declined to consider operative treatment and was given a truss, so that the anatomical state of the parts could not be verified by dissection, though I was quite satisfied that the condition was as I have described.

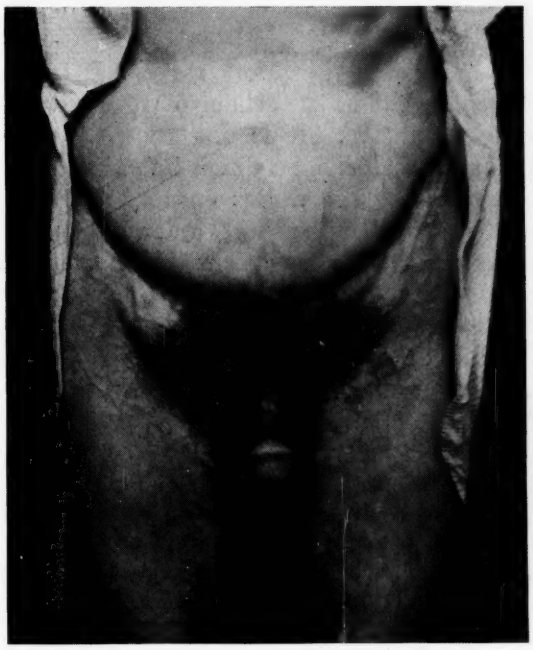

FIG. 21.—Acquired bilateral prevascular femoral hernia.

The distinction between congenital and acquired prevascular femoral hernia has not, as far as I am aware, been clearly made before. Minor degrees of the acquired condition are probably not so very uncommon, though among many thousand patients seen by me at the City of London Truss Society during the last twelve years none has been so well marked as that illustrated in the photograph of *Case* 2. It is probable that this condition would not be amenable to operation, though the congenital variety is curable by a suitable operation as demonstrated in the first case.

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RECONSTRUCTION OF FOREARM AFTER LOSS OF RADIUS.

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William A., age 6½ years, fell in the school playground one day in February, 1921, injuring his right forearm. It is not known if he sustained an open fracture of the radius and so received a direct infection of the bone, but certain it is that an osteomyelitis of the radius ensued, and on July 15 an X-ray examination revealed necrosis of the shaft, and some sequestra were removed through sinuses. By Aug. 28, the patient being still under treatment at the School Clinic, the whole shaft of the radius lay exposed in the depths of an open suppurating wound. He was then referred to Dr. Holland, of Swindon, who transferred the case on Sept. 10 to the Bristol General Hospital, where on Oct. 15 a large sequestrum, comprising the greater part of the shaft of the right radius, was removed.

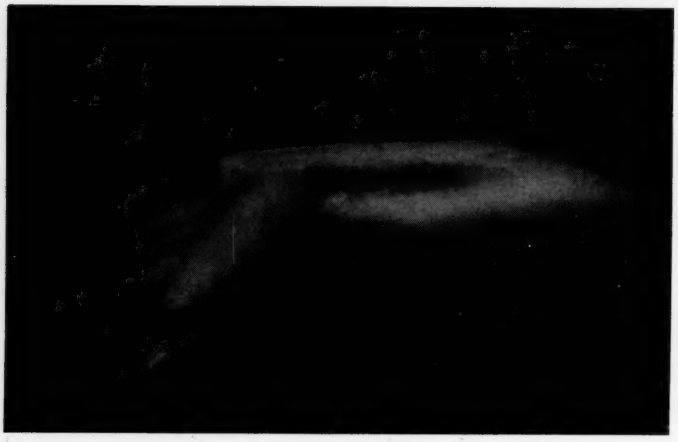

Fig. 22.—Condition of right hand and forearm on Nov. 3, 1922, before operation.

The case was referred to me on Aug. 14, 1922. There were still several discharging sinuses, which finally healed by Nov. 3. There remained only part of the lower epiphysis and a long slender portion of the upper end of the bone, which appeared from an X-ray to be sclerosed and unpromising as a bed for the reception of a bone-graft. The large gap was filled by a mass of dense fibrous tissue, and most of the overlying skin of the back of the forearm was converted into scar tissue. The hand was very helpless, being deflected to the radial side, partly by lack of support of the hand by the defective radius, and partly by contraction of the dense scar tissue (*Fig.* 22).

As a prelude to the transplanting of a graft from the rib, a large tubular skin-graft was taken from the abdomen after removing the scar tissue between the ends of the radius, and this was soundly healed in position on the back of the forearm by Dec. 19, 1922.

When the time for transplanting the bone-graft arrived (April 19, 1923), the reluctant patient, now 9 years old, protested vigorously and did not again present himself at the hospital until early in 1925. The forearm was

again X-rayed by Dr. Harrison Orton, who reported that the poor quality of the upper fragment precluded any likelihood of a successful bone-graft being achieved. Mr. Hey Groves,[1] moreover, had described and illustrated a somewhat similar case, in which two successive attempts at bone-grafting had failed. On these considerations grafting was abandoned and it was decided to adopt a device similar to that employed by Mr. Hey Groves, in order to lessen the serious disability, more especially as the parents (and the boy) were wearied of operations and desired finality.

Operation (May 5, 1925).—A rectangular incision through the skin was made across the line of the wrist-joint and up the forearm along the ulnar border for about 3 in. (*Fig.* 23, A B C). The triangular flap so marked out was raised, the tendons retracted, and the bones exposed.

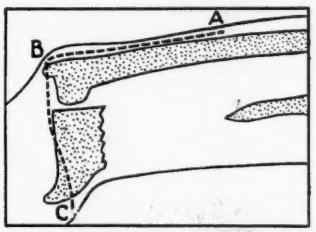

Fig. 23.—Diagram showing bones before operation and line of incision (A B C) through the skin.

In the case previously referred to (the only other case I have been able to find recorded), the ulna was cut through close to the distal radio-ulnar joint and the lower end of the ulna with its styloid process, was removed. The upper fragment was then trimmed to a conical shape and fixed into a hollow scooped out of the upper surface of the distal portion of the radius. In my case a variant of this method was adopted. Out of the upper surface of the lower fragment of the radius a wedge-shaped piece was cut. The ulna was then divided obliquely from above downwards and outwards some 2 in. above the level of the wrist-joint (*Fig.* 24); the pointed lower end of the upper fragment fitted into the V-shaped cavity in the radial epiphysis, whilst the lower fragment of the severed ulna was fixed against the shaft of the ulna by a bone-peg, square in section (P), thus acting as a buttress. In this way the integrity of the wrist-joint was not violated; the resulting joint is perhaps more stable and possibly more mobile.

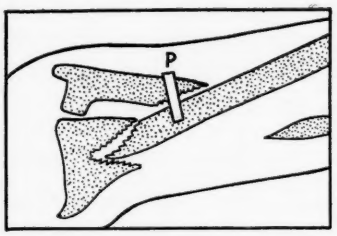

Fig. 24.—Diagram showing position of bones after completion of operation. P, Bone-peg.

In *Fig.* 25 the condition of the bones of the forearm at the present time (March 8, 1932) is seen, and *Fig.* 26 shows the appearance of the limb. The ulna measured from olecranon to styloid process is 7 in. long, whereas that of the other forearm measures 11 in. It is evident that little growth has taken place at the epiphysial line, and this confirms the opinion that an attempt at bone-grafting would probably fail; even if successful, there would have resulted considerable disparity in length between the radius and ulna.

The patient now has a strong forearm with a good grip, free flexion and extension, and a small degree of pronation and supination, so that he is able to lift heavy weights and do laborious manual work. In the X-ray photograph (*Fig.* 25) it will be seen how effectively the buttress strengthens the joint.

COMMENTS.

1. The loss of the shaft of the right radius by necrosis caused a major disability for which the only solution seems to be that adopted in this case.

FIG. 25.—Condition of bones of forearm in March, 1932.

2. The injury was sustained in a school playground and the case remained under the care of the School Clinic for about seven months. This illustrates the wide latitude assumed by Medical Officers of Health nowadays in the treatment of serious injuries.

3. The serious disability tended to increase with the approach of adolescence. The successful grafting of the radius would have failed to correct this, since the capacity for growth in the distal radial epiphysis would have prevented the radius keeping pace with the ulna. The method adopted would appear to be the only practical solution of the problem.

4. The shaft of the ulna has increased in thickness to about double the size of the normal bone.

5. Using the lower portion of the ulna as a buttress, instead of removing it as was done in the case previously recorded, seems likely to give greater stability to the wrist-joint.

FIG. 26.—Appearance of limb in March, 1932.

6. The rarefaction of the carpal and metacarpal bones seen in an X-ray photograph taken in 1925 has disappeared by March, 1932, and is evidence of the increased use of the limbs.

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A NOTE ON THE MOVEMENTS OF THE SHOULDER-JOINT.

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During a recent investigation into the mechanism of the movements occurring at the shoulder-joint some new facts about these movements became apparent and appear to be worthy of record. No reference could be found in any of the anatomical text-books or journals to some of the observations which appear to have considerable practical importance in connection with common injuries and diseases of the joint.

The movements of the shoulder-joint, as described in recognized text-books of anatomy, are: flexion and extension, abduction and adduction, circumduction, and finally rotation. By flexion is meant a carrying of the arm forwards and then upwards, the arm being parallel to the median plane of the body at all stages of the movement. By abduction is meant a carrying of the arm outwards and then upwards, the arm being first parallel to the median plane, then at right angles to it, and finally parallel to it once more. Circumduction and rotation do not need any explanation. In dealing with the actual carrying out of these movements the text-books emphasize the important part played by rotation of the scapula and elevation of the clavicle, especially in flexion and abduction of the arm. Both these movements are carried out not solely at the humero-scapular joint, but are due to a considerable extent to movements at the sterno-clavicular and acromio-clavicular joints. This applies especially to the later stages of the movements of full flexion and full abduction. It is often stated, as, for example, in *Quain's Anatomy*[1] and many other text-books, that the amount of abduction which can take place at the humero-scapular joint is limited to carrying the arm outwards till it is at 64° to the median plane, abduction beyond this point being effected entirely by rotation of the scapula. It has been shown, however, that some rotation of the scapula occurs from the very beginning of either abduction or flexion, and some movement at the humero-scapular joint continues till the arm is fully upright.[2]

From the descriptions given in the text-books, therefore, it would appear that flexion of the arm and abduction of the arm are very different movements. In the case of abduction the arm is carried out till it is at right angles to the median plane. The head of the humerus has therefore been rotated round an axis passing horizontally from before backwards, the greater tuberosity of the humerus has been brought almost against the acromion of the scapula, and the articular surface of the head of the humerus looks downwards, slightly medially, and slightly backwards. Any further abduction that may be carried out is due mainly to rotation of the scapula, but, as already stated, some further movement occurs at the humero-scapular joint.

In full abduction, therefore, the articular surface of the head of the humerus must look almost directly laterally, and be only very slightly in contact with the articular surface of the glenoid cavity of the scapula. In the case of flexion, on the other hand, the head of the humerus is rotated round an axis passing in a transverse horizontal direction; the articular surface of the head of the humerus, therefore, remains in contact with the surface of the glenoid cavity, and in full flexion the head of the humerus looks medially and slightly forwards. Of course in flexion, as in abduction, rotation of the scapula and elevation of the clavicle take place.

The above, I think, is a fair account of the movements of this joint as given in current anatomical literature. With the movements occurring at the sterno-clavicular and acromio-clavicular joints this paper is not concerned, as the text-book descriptions of these movements appear to be complete.

Now it is obvious that we can carry our arms straight up over our heads by two methods, either by flexion or by abduction. From the text-book description of these movements we should infer that the end-result in each case would be different. Observation, however, will show us that the end-results in both cases are identical. If we carry our arms straight upwards it makes no difference, as regards the final position of the bones, whether we do so by flexion or abduction. This fact can easily be verified. With the arm hanging downwards by the side the lateral epicondyle of the humerus is directed forwards and laterally. If the arm is now carried vertically upwards by flexing the limb it will be found that the lateral epicondyle is directed backwards and slightly laterally—in other words, the head of the humerus has, as already stated, rotated round an axis passing horizontally from side to side. If now the arm is again placed by the side and then carried vertically upwards by abduction it will be found that the lateral epicondyle is in exactly the same position as it was when the arm was carried upwards by flexion. So obviously in the case of abduction something more than rotation of the head of the humerus round a horizontal antero-posterior axis has taken place, and the description of abduction in the anatomical text-books appears to be incomplete.

If, once again, the arm is placed hanging down by the side of the body and then carried slowly upwards by abduction, while the position of the lateral epicondyle of the humerus is noted at all stages of the movement, it will be found that, as the arm is abducted, the humerus is rotated laterally round its own long axis. This lateral rotation of the humerus during abduction of the arm is the fact to which I desire to direct attention, for, as far as I can find, it has not been described or noticed in any account of the movements of the shoulder-joint.*

Another method of demonstrating the lateral rotation of the humerus during abduction of the arm is as follows: Place both arms hanging down beside the body, flex the elbow-joint till the forearm is at right angles to

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*Since this paper was written my attention has been drawn to *Applied Anatomy and Kinesiology* by Bowen and McKenzie, published by Lea & Febiger, of Philadelphia and New York, 1919. On p. 101 of this work there is a passing reference to the fact that the humerus rotates laterally during abduction of the arm. No details of the movement are given.

the arm so that the forearm points directly forwards, then abduct the arm, keeping the elbow flexed and the forearm pointing forwards all the time. It will be found that the arms can only be abducted to a little over the horizontal position even though the scapula itself has been raised as much as possible. The reason for this limitation of abduction is that, by keeping the forearm pointing forwards while the elbow-joint is flexed to a right angle,

FIG. 27.—Arms abducted as far as possible while lateral rotation of the humerus is prevented by elbow being flexed to a right angle and forearm being kept pointing forwards.

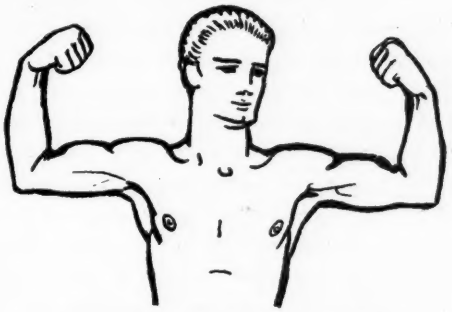

FIG. 28.—Arms in same position as in *Fig.* 27, but humeri turned laterally by turning forearms so that they point upwards.

we have stopped, or at least largely diminished, the lateral rotation of the humerus. If we now, still keeping the elbow flexed, rotate the humerus laterally so that the forearm points upwards instead of forwards we shall find that the arm can be fully abducted till it is directly vertical. (*Figs.* 27–29.) In applying this test it is better to use both arms at the same time, otherwise abduction of one arm may be assisted by tilting of the thorax and the lateral rotation of the humerus obscured. Again, it is obvious that with the arm fully abducted we can flex the forearm inwards over the head, but we cannot flex it outwards; likewise with the arm hanging at rest beside the body we can flex the forearm inwards but not outwards. Of course, owing to rotation of the humerus, we can vary these positions to a certain degree, but still the general direction in which the forearm can be flexed is inwards towards the body, whether the arm is hanging by the side or is vertically upwards in full abduction. Here again it is clear that if rotation of the humerus during abduction had not taken place, then, when the arm is fully abducted, we should be able to flex the arm outwards.

FIG. 29.—Showing full abduction, rendered possible by lateral rotation of humeri.

The plates published by Lockhart[2] show this lateral rotation of the humerus during abduction very clearly, but the fact of the rotation was apparently not observed by him. He draws attention to the fact that abduction of the arm in its later stages—that is, from the horizontal position to when it is vertically upright—is not accomplished wholly by rotation of the

scapula but is attended by considerable movement at the humero-scapular joint; but he does not remark that a very conspicuous part of this movement consists of lateral rotation of the humerus. Fick[3] published a very full account of the movement of the joints, but I cannot find that he noticed this feature of abduction of the arm. I need not enumerate the modern text-books of anatomy that have been searched in vain for any reference to this feature.

This rotation of the humerus during abduction also serves to explain the mechanism of dislocation of the shoulder-joint. The majority of these dislocations appear to be due to sudden hyperabduction of the arm while lateral rotation of the humerus is prevented, either by the suddenness of the movement or by contraction of the muscles passing from the thorax to the anterior aspect of the humerus. This latter contingency arises when, with the forearm flexed to a right angle with the arm, a man falls sideways and the volar aspect of the hand and forearm comes in contact with the ground. In the effort to save himself he contracts all the muscles passing from the thorax to the arm, including the teres major, latissimus dorsi, and pectoralis major, all of which are inserted into the anterior aspect of the humerus, and, while the impact of the fall forces the arm up into the position of full abduction, the contraction of these muscles prevents lateral rotation of the humerus.

Taking into account the fact that so-called abduction of the arm is a very complicated movement involving (1) true abduction—that is, a carrying of the arm away from the body in a lateral direction, (2) lateral rotation of the humerus, and (3) rotation of the scapula and elevation of the clavicle, we can see how it comes about that the position of the bones at the end of abduction is exactly the same as if the arm was carried to the vertically upright position by flexion. It would also appear that great care should be exercised in examining cases of limitation of movement of the shoulder-joint, for, if this is not done, many such cases may be wrongly diagnosed as impairment of the power to abduct, whereas the real impairment is the inability to rotate. In fact, it is also possible that some of the splints and arrangements used in cases of injury in the vicinity of the shoulder-joint may be ill advised because they do not allow for this lateral rotation; and, from what has been written above, it is apparent that if the power to rotate the humerus is lost, abduction of the arm must be gravely affected. In this respect it is of interest to note that Jones and Lovett and other orthopædic surgeons advocate putting up all such injuries with the arm fully rotated laterally so that the forearm points upwards when the elbow is flexed to a right angle.[4] They thus recognize that this position gives the best end-results, but they apparently do not recognize the reason for this fact.

In abducting the arm the muscles which contract are the supraspinatus, deltoideus, and trapezius. Other muscles in the neighbourhood are relaxed. The lateral rotation of the humerus during abduction of the arm is apparently brought about by the muscles which are contracting, and of these the most important in producing the rotation of the humerus are the posterior fibres of the deltoideus which take origin from the lower lip of the crest of the spine of the scapula and are inserted into the deltoid eminence on the humerus. The rotation is aided by the fact that the deltoid eminence of

the humerus is slightly more on the anterior than the posterior aspect of the bone. The supraspinatus may help in procuring this rotation, as it arises slightly behind the shoulder-joint and is inserted directly lateral to it. But in the latter half of the movement of abduction the rotation of the humerus is mainly due to the position of the scapula and the outline of the lateral edge and under surface of the acromion and coraco-acromial ligament. These two structures form an arch over the glenoid cavity of the scapula, but it should be noted that this arch is nearer to the glenoid cavity in front than it is behind. It should also be noted that though some rotation of the humerus takes place during abduction of the arm from the side to the horizontal position, most of it occurs during the latter half of abduction when the arm is carried from the horizontal position to the vertically upright one, and it is during this later stage that the greater tuberosity of the humerus comes in contact with the lateral edge and under surface of the acromion and coraco-acromial ligament. As the arch formed by these two structures is farther from the glenoid cavity behind than it is in front, the greater tuberosity of the humerus slides backwards when it comes in contact with them and thus rotates the humerus laterally. Finally, the greater tuberosity of the humerus is directed straight backwards and the shaft of the humerus occupies the interval between the acromion and the coracoid process. The infraspinatus and teres minor might from their position appear to help in rotating the humerus laterally during abduction, but they are relaxed in abducting the arm, and in addition their tendency to rotate the humerus laterally is outbalanced by the tendency of the pectoralis major, teres major, latissimus dorsi, and subscapularis to rotate the humerus medially. It is evident, therefore, that all the muscles which are relaxed in the act of abduction have little influence on the rotation of the humerus.

In abducting the arm we contract one set of muscles and stretch another, and if we use the arm as a tensile organ and hang by it from a beam it is clear that the tensile strain of the body weight will be transmitted through the set of muscles that have been stretched. These stretched muscles are the pectoralis major, the teres major, and the latissimus dorsi, all of which are inserted into the anterior aspect of the humerus. But if the humerus is rotated laterally during abduction it would appear that all these muscles have undergone a good deal of torsion, and we should expect that if a man was hanging by one arm his body would swing round till the torsion was undone. This is what actually happens. Suspended by one arm the body will not come to rest till it has rotated round the humerus so as to undo the lateral rotation of this bone and the torsion of these muscles. The arm is then in a position of true abduction combined with considerable tilting of the thorax. This is an experiment that anyone can try on himself. Of course if the arm is carried up by flexion similar torsion of the muscles occurs.

SUMMARY.

Abduction of the arm is a complicated movement consisting of true abduction of the humerus at the humero-scapular joint combined with rotation of the scapula and elevation of the clavicle. But, in addition to these

factors, abduction of the arm is always accompanied by lateral rotation of the humerus to an extent of over 90°. This rotation takes place for the most part during the later stages of abduction when the arm is raised from the horizontal position until it is vertically upright, but a certain amount of it occurs during the first half of abduction. It is on account of this lateral rotation of the humerus during abduction that the position of the humerus at the end of full abduction is exactly the same as it is at the end of full flexion. This lateral rotation of the humerus during abduction of the arm has not, as far as I can find, been described in any publication previous to this paper.

I desire to thank Miss O'Brien for drawing the illustrations for this paper.

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SOME EXPERIENCES WITH SYMPATHETIC GANGLIONECTOMY.

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The sympathetic nervous system is rapidly coming to the forefront of surgical interest, and both Lord Moynihan, in this country, and W. J. Mayo, in America, have expressed the opinion that much in the future development of surgery will lie in the direction of the autonomic nervous system. A more complete understanding of its anatomy and physiology is still wanting, though much has already emerged from the abundance of research which is being pursued in most countries at the present time. It is a matter of no small satisfaction to realize that the pioneer work in this sphere came from the British school of physiology, the names of Gaskell and Langley being pre-eminent; indeed, Albert Kuntz,[1] in introducing his book, *The Autonomic Nervous System*, a complete résumé of all our knowledge of this subject up to 1929, says of the latter, "the term autonomic nervous system ought to be used in recognition of Langley's great contribution to our knowledge."

The autonomic nervous system is divisible into two antagonistic parts—the parasympathetic and the sympathetic, and it is to the latter that surgical procedures have been directed. No attempt will be made here to discuss all that is known of the physiology of the whole autonomic system, though a little must be said of those recognized functions of the sympathetic part of the system which surgeons have attempted to modify in some way or other by operative interference; it will be evident, too, how these surgical experiences have stimulated further research and have led to fresh knowledge.

It has long been known that stimulation of the nerve-fibres lying within the adventitia of an artery leads to spasm of that artery, and Leriche, seeing the practical application of this fact, evolved his operation of peri-arterial sympathectomy. This he used for vasospastic conditions of the legs and arms, particularly Raynaud's disease and Buerger's disease (thrombo-angiitis obliterans). He also applied it to such maladies as chronic varicose or traumatic ulceration of the legs, delayed union of fractures, and even to traumatic tuberculosis of joints, conditions, he argued, wherein any improvement in circulation would seem to promise a stimulus to the sluggish processes of healing. This vasodilatation—for that is what the operation was calculated to produce—is found to be of temporary duration only,[2] lasting about five to six weeks, after which time the arteries would appear to regain their normal tone, so that, though of considerable help in the more local conditions such as the three last mentioned, in which a fuller blood-supply of a few weeks' duration is sufficient to initiate a more vigorous healing reaction, the results are disappointing in the more generalized states such as Raynaud's and Buerger's

diseases, in which the improvement in circulation needs to be lasting before permanent alleviation can be expected; the conception was good but the anatomy was incompletely understood.

At about the same time as Leriche was studying these problems of vasospastic diseases other workers were attempting to discover some procedure by which the pain of angina pectoris could be permanently controlled. The names associated with these researches were Jaboulay, Jonnesco, and Danielopolu in Europe, and Coffey, Brown, and Cutler in America. They attacked different parts of the cervical sympathetic chain on the left side on the assumption that the pain of angina is due to spasm of the coronary arteries and aorta and that the impulses are conveyed through the afferent components of the sympathetic cardiac nerves. The surgical treatment of angina pectoris has never been popular with British cardiologists, as they consider the pain a useful danger signal and the operations suggested too severe for patients suffering from cardiac lesions. However, as with the problems of vasospastic diseases, this work has stimulated further research.

Quite a different aspect of the sympathetic part of the autonomic nervous system has received attention in Australia, and in 1923 Hunter published his brilliant researches on the dual innervation of the voluntary muscles, and Royle described his equally brilliantly conceived surgical approach to certain spastic conditions of the muscles, based on these researches. He severed the grey rami communicantes of the cervical or lumbar ganglia, according to whether the spasticity involved the arms or legs respectively, with the idea of cutting out the overactive plastic component of muscular tone. His patients, for the most part, were wounded soldiers with spinal injuries—patients, in other words, who formerly had been normal in body and mind, and who, therefore, would co-operate intelligently and enthusiastically in the re-educative muscular exercises which are almost the most important part of treatment. Royle reported many successes, but the operation has not been so encouraging in the hands of others; and Hunter's researches have as yet remained unconfirmed. In civil practice so many of these cases occur in children who have sustained birth injuries, whose mentality is defective, and whose powers of co-operation are distinctly poor. Probably this fact accounts for the failures experienced by many surgeons who have attempted to deal with spastic diplegias. In this country the most that is attempted from a surgical point of view is some modification of Stoffel's operation, and then only if the child exhibits considerable intelligence. Nevertheless, the work of Hunter and Royle aroused universal interest, and observers realized that the varying results of all these different operations were due to an inadequate knowledge of the anatomy of the sympathetic system.

I do not propose to recount in detail the more recent anatomical researches into this problem; suffice it to say that the work of Hovelacque, Delmas, Latarjet, and Langley in Europe, and of Kuntz, Ranson, and Learmonth in America, has demonstrated the following facts:—

1. The nerve-supply to the arteries of the limbs is somatic, not visceral—that is, the nerves lying within the adventitia of the main artery as it enters the limb do not comprise the total nerve-supply of the entire arterial tree distal to this point; there is a constant accession of fibres derived from the

nerves to the voluntary muscles as one passes distally. From a practical point of view it can be said that the nerves lying within the adventitia of the femoral artery just distal to Poupart's ligament supply the artery and its branches as far as the level of the knee and no farther; everything below this derives its nerve-supply from fibres entering the vessels at lower levels, e.g., a branch from the nerve to the popliteus can always be found to enter the anterior and posterior tibial arteries. In like manner fibres within the adventitia of the third part of the axillary artery supply the tree only so far as the elbow. It is therefore obvious that in order to denervate the whole arterial tree of a limb one must attack the source.

2. Complete sympathetic denervation of the vessels of the lower extremity can only be achieved by removal of the 2nd, 3rd, and 4th lumbar ganglia with the intervening chain on the appropriate side. Complete sympathetic denervation of the vessels of the upper extremities demands extirpation of the inferior cervical ganglion, the 1st and 2nd dorsal ganglia, and the intervening chain. The 2nd dorsal ganglion is of importance, as in over 20 per cent of cases a large branch is found to pass from it into the brachial plexus (Kuntz). Neglect of this may lead to disappointing results.

No account, however brief, would be complete without mentioning the very convincing experiments carried out by Horton and others at the Mayo Clinic. A number of dogs were taken and in one series a peri-arterial sympathectomy was performed in Scarpa's triangle on one side; in a second series the 2nd, 3rd, and 4th lumbar ganglia and intervening chain were removed on the same side as in the first series; a third series was taken as a control. It was arranged that the second part of the experiment should be done by a separate group of workers, who were not informed as to the exact details of what had been done in each animal; and in order to make the investigation more perfect the same incisions were made in each dog. Thus, in the first series, although the operation was performed through an incision in the upper part of the thigh, a second superficial mid-line abdominal incision was also made; in the second series, although the operation was performed through a mid-line abdominal incision, a second superficial incision was made in the upper part of the thigh; and in the third series two similar but superficial incisions were made as in the other two series. The experiments were, therefore, perfectly controlled. The second stage of the experiment was carried out about six to eight weeks later, and consisted of opening the abdomen of each anæsthetized dog and injecting mercury at systolic pressure into the abdominal aorta. X-ray pictures of the lower extremities were then taken before death. In the first series the pictures showed dilatation of the arterial tree on the side of operation as far as the level of the knee but no farther; the second series showed dilatation of the whole tree, right down to the tips of the toes, as compared with the untouched side; the third series showed no difference. The results fulfilled exactly the anticipations of the first group of workers.

3. The circular fibres of the sigmoid colon are supplied by a definite branch of the presacral nerve of Hovelacque, known as the 'lumbar colonic nerve'; this has recently been confirmed by Learmonth and Markowitz.[3] Excision of the presacral nerve, which is really a plexus, releases the

sympathetic control of the sigmoid, and has been used for certain cases of spastic colon, of which one case is recounted below.

The Conditions for which the Operations have been Advocated.—Experience has shown that the following conditions are amenable to treatment by sympathetic ganglionectomy, but of course all other forms of treatment should be tried before recourse is had to these operations.

1. *Raynaud's Disease.*—This disease, excepting in its most advanced stages, would seem to be due entirely to overaction of the sympathetic nerve-supply; at any rate, release from sympathetic control checks the arteriolar spasms and thereby leads to a cure.

2. *Thrombo-angiitis Obliterans.*—In this disease there are two factors at work—namely, the inflammatory lesion of the vessels resulting in narrowing of the lumen, and a superimposed spasm. If this latter factor is of sufficient degree, the operation of sympathectomy will result in great improvement—even to checking a threatened gangrene. Whether or not a case is suitable for operation is ascertained in the following manner. A laboratory thermometer is bandaged to the affected limb in such a manner that the bulb lies between, and in contact with, two fingers or toes, as the case may be. The temperature is noted, and that of the mouth is also taken. An intravenous injection of 50 millions T.A.B. is then given and the highest subsequent temperatures in both mouth and limb are recorded. The temperature of the limb before the injection may be little above room temperature, and any rise during the fever stage is due to vasodilatation consequent on relaxation of sympathetic tone. Brown, at the Mayo Clinic, has found from experience that if the difference in degrees between the rise of temperature in the mouth and that of the limb is three times the rise in the mouth, then the case is suitable for operation. Thus, if the rise of temperature in the mouth be 3° F. and that in the limb be 16° F., the index becomes $\frac{16-3}{3} = 4{\cdot}3$, and the patient would receive great benefit. If the index is below 3 it indicates that the thrombosis is responsible for the trophic changes to a much greater degree than the spasm. In Raynaud's disease the index may be as high as 10 to 20.

3. *Certain Cases of Severe Constipation* due to spasm of the colon are much benefited by removal of the presacral nerve, since the sympathetic not only innervates the circular fibres and sphincters but also inhibits peristalsis.

4. *A Miscellany of Cases exhibiting Minor Trophic Changes*, wherein are present signs of sympathetic overaction in the form of coldness, blueness, clamminess, shininess of the skin, etc. Of such conditions may be mentioned causalgia of amputation stumps, some types of scleroderma, indolent ulcerations. In this category should be included also certain types of peri-arthritis, and although the writer has had no personal cases so far, yet from his experience as assistant to Dr. A. W. Adson at the Mayo Clinic he can speak favourably of this method of treatment in carefully selected cases. The necessary features of a suitable case are that there should be no bone changes, that there should be evidence of arterial spasm, and that the joints affected should be peripheral ones. After all, sympathectomy in these circumstances establishes permanently that which many forms of conservative treatment such as radiant heat, diathermy, whirlpool baths, etc., merely succeed in

producing temporarily—namely, vasodilatation; but unless there is quite definite evidence of sympathetic overaction of a degree sufficient to give an index of 3 or over in Brown's test, nothing but disappointment will ensue and the method of treatment will be brought into disrepute.

5. *Certain Types of Pain*, thought to be due to vascular spasm, though not accompanied by indisputable evidence of sympatheticotonia. The anatomical basis for operation in these cases is not entirely established, but some authorities maintain that afferent fibres from the arteries carry painful sensations up to the brain and that sympathetic ganglionectomy, by severing the pathway, will relieve the pain. Certainly, the pain of Raynaud's and Buerger's diseases is abolished by the procedure, but in these conditions the spasms are of such severity that trophic changes develop, whereas in the cases under the present heading the nutrition of the part affected would not appear to be impaired. One case of intermittent claudication is reported below, and, as will be seen, the result was disappointing. Leriche and Bornard have used different forms of retroperitoneal sympathectomy for the type of dysmenorrhœa said to be due to fibrocystic ovaries and have reported successes, but the writer has no experience in this field. As already said the treatment of the pain of angina pectoris by any form of operation is viewed with disfavour in this country.

Choice of Cases.—The operations of dorso-cervical sympathetic ganglionectomy and of lumbar sympathetic ganglionectomy are more severe procedures than Leriche's peri-arterial sympathectomy, and in elderly, enfeebled, toxic patients should not be attempted; the lumbar operation is contra-indicated in obese subjects. In such cases a peri-arterial sympathectomy might, with advantage, be undertaken; but, as already pointed out, it is anatomically incomplete, and when possible the full sympathectomy should be performed.

Operations.—Both operations have been illustrated and described in detail by Adson[4, 5, 6, 7] on more than one occasion, and except for minor differences in technique the following cases were performed by his method, the essential points of which are briefly as follows. The operation for the removal of the inferior cervical and 1st and 2nd dorsal ganglia is based on Henry's exposure of the cervico-dorsal ganglion. The transverse process of the first or second dorsal vertebra and the head and neck of the corresponding rib are removed, and the ganglia are found lying in front, on the pleura. The inferior cervical ganglion lies on the neck of the 1st rib, and the 1st dorsal ganglion is often fused with it. The 2nd dorsal ganglion lies in front of the head of the 2nd rib. The grey and white rami communicantes are seen to enter and leave the ganglia in radiating fashion, and the intervening chain is as thick as the radial nerve, though this is variable. Sometimes, if there has been old apical inflammation, the ganglia and chain are bound by adhesions. If both sides are to be treated, they can be reached by a single dorsal mid-line incision; if only one side requires operation, the incision is made 1 in. lateral to the mid-line. The patient is placed in the laminectomy position for the operation, and an illuminated spatula is essential for the deeper dissection. The dead space resulting from the removal of bone should be obliterated so far as is possible. The patient should be nursed on the back with a pillow between the shoulders.

The 2nd, 3rd, and 4th lumbar ganglia lie on the sides of the bodies of the corresponding vertebræ. They are behind the vena cava on the right side, and the aorta on the left. If it is wished to remove the chain of ganglia on both sides, the operation is best performed through a mid-line abdominal incision with the patient in the Trendelenburg position. On the right side the ganglia are approached through an incision through the posterior abdominal peritoneum, internal to the cæcum and below the root of the mesentery. On the left side it is necessary to incise the peritoneum lateral to the descending colon and to reflect the colon towards the mid-line. Again, an illuminated spatula or a head-light is essential. If it is intended to attack one side only, this can be done retroperitoneally through a lumbar incision as for exposure of the ureter.

The presacral nerve lies in front of the 5th lumbar vertebra in the angle of the bifurcation of the aorta, and the lumbar-colonic nerve runs with the inferior mesenteric artery: both are most satisfactorily removed transperitoneally.

CASE REPORTS.

The following are a few examples of cases treated by one or other of these operations. In each case many forms of conservative treatment had been given first but without benefit. The writer feels that sufficient time has elapsed in each case since operation to merit the result being considered satisfactory. They were reviewed in April, 1932, and each one was found to have maintained the original improvement.

Case 1.—J. S., age 21, female. Hairdresser.

Admitted to hospital in February, 1930, complaining of increasing numbness and tingling of the fingers of both hands. This trouble had first been noticed about twelve months before, during the winter. As the warmer weather came on she became more comfortable, but as soon as the present winter started her fingers became cold, blue, tingling, and numb whenever the hands were exposed even for short periods to cold. Latterly the fingers had started 'going dead', and she began to find her work as a hairdresser increasingly difficult, so much so that she feared she would have to give it up. There were no past illnesses.

The girl was somewhat pale and fat, with a small goitre and sluggish peripheral circulation. The fingers and hands were cold, blue, and moist, but until she held them in cold water the fingers did not go 'dead', and after recovering they became congested and throbbing. The pulses were full and soft. Her feet were also cold, but did not cause any discomfort, and exhibited no vascular spasms or trophic changes. General examination otherwise revealed nothing abnormal. There was no radiographic evidence of cervical rib. A diagnosis of Raynaud's disease was made.

The patient failed to respond to various forms of physiotherapy, and it was decided to perform a bilateral cervico-dorsal sympathetic ganglionectomy. Unfortunately the pleura was torn on the second side, and she developed pneumonia. However, she recovered from this and the hands and fingers have remained warm and dry ever since. She is doing her work as a hairdresser, which entails using cold water sometimes, but this gives no trouble. She has a Horner's syndrome on both sides, as always happens when the inferior cervical ganglion is completely removed.

Pathological Report.—Professor Haswell Wilson says of the section of the tissue removed: "This shows diffuse fibrosis and degenerative changes in the ganglion cells."

Case 2.—V.B., age 27, male. Copper-smith.

Admitted to hospital in August, 1930, complaining of cramp-like pain in the right calf. This began two years ago, within a fortnight of appendicectomy. At

first he thought it was weakness from the enforced lying in bed, but even when he had otherwise returned to good health and had restarted work this pain came on about 5 o'clock every afternoon. It became gradually worse and started earlier in the day, and at the present time it begins as soon as he is out of bed, and after walking only a quarter of a mile he has to stop to rest. He gave up his work several weeks ago. The pain is of a 'crampy nature' and he says the foot sometimes feels numb. There is never any pain in the left leg.

The patient is a strong healthy man with no history of any serious illnesses apart from appendicitis two years ago. He is not a heavy smoker. There is no muscular wasting, but the right foot feels colder than the left, it is somewhat dusky in colour, and the dorsalis pedis and posterior tibial pulses are only about half the volumes of those on the left side. General examination does not reveal any other abnormality.

A diagnosis of thrombo-angiitis obliterans was made, and, conservative treatment having failed, it was decided to perform a lumbar sympathetic ganglionectomy on the right side. This was done transperitoneally. The man's progress was rapid and he claimed to feel more comfortable in the right calf. For the first week or so after getting up from bed he experienced a slight cramp, but this soon disappeared and he returned to work.

On examining him in April, 1932, he stated that he was back at full work and never under any circumstances suffered any pain whatsoever. Both legs appeared exactly similar, though the right one is the warmer, and the pulses on the right side were as full as those on the left. He says he never perspires in the right leg, and this is reflected in the condition of the insides of the shoes, the right one being dry and shiny.

Pathological Report.—The report on the section of the tissue removed, made by Dr. F. W. Mason Lamb, states that, " The ganglion cells show degeneration. Many have lost their nuclei. There are small areas of hæmorrhage in the connective tissue ".

Case 3.—J. P., age 30, male. Turner.

Admitted to hospital in August, 1930, complaining of extreme tenderness of the amputation stump of his right thumb. The thumb was crushed in a lathe in June, 1929. It was trimmed in the casualty department, but because of sepsis an amputation was performed. This became painful because of an adherent scar, and a second amputation was done. The stump was still tender and painful and the removal of amputation neuromata in no way improved his condition. I saw him for the first time in May, 1930. The stump was then so sensitive that if he caught it against his clothing he gave a shudder of pain ; he was completely incapacitated.

On examination it was seen that the stump was blue, cold, clammy, and the skin was shiny and atrophic. This condition also involved the ball of the thumb. It appeared to be a true case of causalgia with very definite evidence of sympathetic over-action. In view of the very localized distribution it was decided to give a course of diathermy and massage, but there was no improvement after two months of this treatment. A cervico-dorsal sympathetic ganglionectomy on the right side was advised, and this was carried out ; the effect was immediate. The stump at once became warm, pink, and dry, and all tenderness and pain disappeared, and within a fortnight the skin had lost its atrophic appearance.

On examination in April, 1932, this healthy and comfortable condition was maintained.

Pathological Report.—The report by Dr. F. W. Mason Lamb states, " The ganglion cells vary very much in size. Some are shrunken and have lost their nuclei. The connective tissue appears to be increased."

Case 4.—S. O., age 41. Housewife.

Admitted in June, 1931, complaining of coldness of both hands, particularly of the right one, and stiffness of the fingers with consequent inability to do such things as sewing and knitting. The coldness commenced about ten years ago, but

was then diagnosed as chilblains; no beneficial treatment was found. There is often a burning throbbing pain in the hands, and when they are warmed they become cyanosed and ache. The condition becomes aggravated in cold weather. The fingers cannot be extended.

On examination both hands are found to be cold and a little moist. The thumb and all fingers on the right side are flexed and cannot be extended voluntarily, though passively they can, i.e., there is no contracture. The movements of the left hand are clumsy, but extension is not limited. There is diminution of all forms of sensation in the right fingers and hand as high as the wrist. Although the patient was kept in bed for the first few days after admission to hospital the hands were at no time observed to become really warm; they became cyanosed when warmth was applied. The radial pulses are small in volume. She is quite unable to perform any of the more complicated movements with the fingers of the right hand. The feet are also always cold but do not cause any serious discomfort.

On injecting 50 million T.A.B. intravenously the whole of the limbs became warm and pink within two hours, and remained so for several hours, and the patient voluntarily declared that she had not had such comfort for many years.

It was decided to remove the stellate and second dorsal sympathetic ganglia with their connections on the right side. There was uninterrupted recovery and the hand remained warm, dry, and pink, and was much more comfortable.

As seen in April, 1932, the comparison of the two hands is very remarkable, for whereas the left hand is still cold, blue, and stiff, the right is of normal colour, it only becomes cool when exposed to cold but never aches or feels stiff, and when warmed in water or by the fire it never 'goes black' like its fellow on the opposite side. Moreover, she can now extend the thumb and index and middle fingers and is able to sew and do many things which she had not done for ten years.

Pathological Report.—The histological report says, "There is some fibrosis of the substance with atrophy of a few of the ganglion cells." (G. H. W.)

This case was thought to be an example of Raynaud's disease, but atypical, in that the spasm appeared absolutely constant. The response to injection of T.A.B. seemed to confirm this, for the pulses became soft and full. An X-ray picture failed to reveal any evidence of a cervical rib, which diagnosis had been considered, it being thought that the patient resembled the cases recently reported by Telford and Stopford. The inability to extend the fingers and the sensory changes were thought to be due to the prolonged vascular change. However, now that the spasm has been released by the operation it is realized that there is an ulnar paralysis present; and the diagnosis is almost certainly one of cervical fibrous band occurring in a patient whose sympathetic nerve-supply to the brachial plexus runs in the lowest trunk of the plexus, as described by Telford and Stopford. She has been advised to have a second operation for this, and has received such benefit from the first operation that she has accepted.

Case 5.—E. B., age 27, female. At home.

Admitted to hospital in early October, 1931, complaining of backache, difficulty in walking, pain and numbness of the hands and feet, and trophic ulcers of the feet, over a period of seven years; excessive perspiration of the legs during the last eighteen months.

The backache and part of the difficulty in walking were due to bilateral congenital dislocation of the hips and bilateral pes cavus, for which conditions she had been operated on at various times by Mr. Naughton Dunn with satisfactory results. During the same time the circulation of the legs in particular, but of the hands, too, became progressively worse, so that the hands and legs were always cold, blue, and numb, and from time to time shallow painful ulcers appeared in the region of the heels and ankles. These always healed with rest in bed, but tended to break down as soon as the patient attempted to get about again. The legs ached a great deal. During the past eighteen months the perspiration in the legs became so copious that her bedclothes were damp each morning. The hands also perspired a great deal, and the fingers tingled and were always cold and blue. These vasospasms never seemed to relax.

On examination the legs are mottled with various shades of blue and red; the capillary circulation is sluggish; the skin is cold, feeling little above room temperature; there is an ulcer 2 in. in diameter on the left heel and another of ½ in. in diameter over the right external malleolus; these ulcers are very tender on the slightest pressure. The bedclothes around the feet are damp from perspiration, which can be seen trickling off the feet and lower halves of the legs. These vascular changes seem to cease above the knees, though the whole body and upper limbs are moist with perspiration. The legs below the knees show an impairment of all forms of sensation and there are patches of complete loss of tactile sensation. The dorsalis pedis arteries cannot be felt, and the posterior tibial arteries give very thin pulses. The vascular spasm has not been observed to relax at all.

Fifty million of T.A.B. intravenously caused a rise of temperature in the mouth from 98° to 100·4° in two hours, and in the feet from 68° to 96° in six hours—a rise of 28°. It was decided to advise a bilateral lumbar sympathectomy.

This was carried out and the patient made an uninterrupted recovery. From the moment she left the table the lower limbs were warm, dry, and pink, and they have remained so ever since. The ulcers had both healed within a week's time. She was allowed up at the end of a fortnight and then walked quite well. She declares that the feet have been more comfortable since operation than for many years, and now asks that something similar may be done for her cold hands.

PATHOLOGICAL REPORT.—The report states, "There is distinct inflammatory cell infiltration and many of the nerve-cells have lost their nuclei and staining reactions." (F. W. M. L.)

Case 6.—N. H., age 22, female. Housework.

I had the opportunity of operating on this patient in collaboration with my senior colleague, Mr. J. B. Leather, under whose care she was admitted.

Admitted to hospital in March, 1931, with the complaint of obstinate constipation over a period of more than seven years. She had had a long history of surgical and medical treatment, of which the following are the main points. Appendicectomy in 1923 for chronic appendicitis. Following this she began to be troubled by constipation, but managed to get regular movements with various aperients. Six months later the tonsils and adenoids were removed. In 1924 she had an illness which she describes as 'ulcerated bladder' and was in hospital for eleven weeks. After this she developed 'stomach trouble' and the constipation became more troublesome. She was taken into another hospital in 1928 where a thorough X-ray examination of the abdomen was carried out but nothing abnormal was found. She was then examined periodically in the out-patient department, and various forms of treatment were advised for the constipation, which was getting worse and which had reached the stage of requiring enemas on alternate days; if these were omitted, she would go for ten days without evacuating the bowel and would then be forced to have recourse to enemas in order to get comfortable again. She was once again admitted to the wards, but no treatment was of any use, and it was decided to resect the presacral and lumbar colonic nerves on the assumption that this was some form of achalasia.

The operation presented no difficulties and the lumbar colonic nerve was found as a sturdy single branch running along with the inferior mesenteric artery. A slender adhesion between the cæcum and middle of the transverse colon was cut and tied off.

The patient began to have easy bowel movements from within a few days of operation. When interviewed in April, 1932, she stated that this satisfactory state of affairs had been maintained, and this without the aid of enemas or laxatives.

It may be argued quite reasonably that this is not a fair case upon which to pass judgement, as a definite adhesion was severed. This adhesion was very slender and about 3 in. long, and it certainly did not cause any kinking of the colon or cæcum. Moreover, since an enema would always produce a good result before operation it can be safely assumed that the contents of the colon passed the area of adhesion without difficulty, and were in fact held up in the pelvis.

PATHOLOGICAL REPORT.—"No abnormality is seen in the ganglion cells, but a collection of lymphocytes is seen in the neighbouring tissues and there is some evidence of chronic inflammation of the connective tissue in the region of the nerve." (F. W. M. L.)

Another case of causalgia, almost exactly the same as *Case* 3, in history, signs and site, was treated by a dorso-cervical sympathectomy. For three weeks the result was perfectly satisfactory, the stump becoming warm, dry, pink, and painless, but the patient then relapsed and evidence of vascular spasm accompanied the return of extreme tenderness. I believe the anatomical basis for this operation to be so sound that I attribute this failure not to faulty theory but to incomplete execution. This particular operation was performed through the second transverse process and rib, and the inferior cervical ganglion was only partially removed; the fact that the patient did not show a Horner's syndrome supports this view. It is hoped that he will submit to a second operation.

I was also persuaded to try this type of treatment for a case of severe intermittent claudication in one leg. The man, age 56, had arteriosclerosis, but there were no vascular spasms observable. He recovered perfectly from the operation, but there was not the slightest improvement as regards pain. This was an early case and the fever test was not applied. Such a result therefore should not be held to condemn the operation; it was an ill-chosen case and incompletely investigated.

I have, more recently, performed one or other of these operations in a number of other cases, and though the results promise well it is as yet too early to claim them as examples of permanent relief.

COMMENTARY.

It is realized that the method of recording surface temperatures by bandaging the bulb of a laboratory thermometer between adjacent fingers or toes is a rough one as compared with those of the electrothermal couple or the calorimeter, but for practical purposes it is sufficiently accurate; for experimental work it most certainly is not.

It is a matter of no small interest that the thickness of the sympathetic cords intervening between ganglia varies in different patients; sometimes they are as thick as the ulnar nerve in the arm; at other times they are no bigger than the posterior interosseous nerve. In some cases there seems to be evidence of old inflammation around them in the form of adhesions, and in *Case* 5 there were signs of recent inflammation in the form of round-celled infiltration. The common finding of changes within the ganglia themselves is of great interest too, and suggests that they may have been the seat of some infective or toxic process. Investigations into these problems are being pursued.

SUMMARY.

1. If it is assumed, as it is by many authorities, that vasospastic conditions are due to overaction of the sympathetic nervous system, then the operation of peri-arterial sympathectomy as an attempt to denervate the whole arterial tree of a limb is anatomically unsound. Nevertheless, Leriche's

operation has its uses in cases of temporary interference with the nutrition of limbs, and may also prove beneficial in those patients requiring full sympathectomy but who are not in a fit state for the major procedures.

2. Complete sympathetic denervation of the upper limb demands removal of the inferior cervical and 1st and 2nd dorsal ganglia together with the intervening chain; that of the lower limb requires removal of the 2nd, 3rd, and 4th lumbar ganglia plus the intervening chain.

3. The sympathetic control to the sigmoid colon can be abolished by excision of the presacral nerve, or by severing the lumbar-colonic nerve.

4. Before deciding upon sympathectomy in vasospastic conditions it must be quite certain that the case presents irrefutable evidence of sympathetic hypertonicity—that is, an upset of the normal sympathetic-parasympathetic balance. This can be proved by inducing a fever in the patient and noting the degree of vasodilatation which ensues.

5. Cases illustrating the employment of this form of treatment are recorded.

6. Comment is made upon the appearance of pathological changes within the ganglia removed.

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ACUTE PERFORATED PEPTIC ULCER.

A REVIEW OF 64 CASES.

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This study was undertaken with a view to finding out, in particular, the late results of acute perforations of peptic ulcers. Incidentally other data were given notice and are recorded below. It is often stated that acute perforation of a gastric or duodenal ulcer, given operation, leads to its complete healing. If this is the case, both doctors and afflicted patients should regard the onset of perforation as a blessing in disguise—provided, of course, that the patient is given a reasonable chance of recovery by early operative treatment. Such a state of affairs seems at best too good to be true, while at least it warrants investigation.

The number of cases investigated was 64. All were operated upon by one of us (J. G.) during the years 1922 to early 1929, so that none has a post-operative history of less than two years, and in 24 cases the period is five years or over.

Sex Incidence.—Of these cases, 58 were males and 6 were females, a proportion, roughly, of 10 to 1. These figures bear out a fact which we are sure is the experience of every surgeon who has dealt with much much emergency surgery—namely, the preponderance of males over females amongst the individuals suffering from this acute abdominal catastrophe. For example, Urrutria[58] records 49 cases in males out of a total of 52, Dineen[13] 138 males in 142 cases, Hinton[27] 103 males in 105 cases, and Dunbar[16] 355 males in 387 cases. Other authors who have recorded a series of cases, are Brown,[5] Kreuter,[30] Platou,[45] Robitshek,[47] and Williams and Walsh,[60] all of whom find sex relativities similar to those recorded above.

Localization.—*Table I* shows the site of the ulcer in the cases of the present series.

Table I.—Localization in 64 Cases of Peptic Ulcer.

	Duodenal	Pyloric	Gastric
Males	52	4	2
Females.. ..	6	—	—
Totals ..	58 (90·7%)	4 (6·2%)	2 (3·1%)

While in most cases of acute perforation the exact site of the ulcer can be recognized with relation to the pylorus, in some the scarring and induration have obliterated the pyloric vein of Mayo or are masking the definition of the thickened muscle constituting the pyloric sphincter, thus making it impossible to identify the site definitely as duodenal or gastric. These latter cases are inserted into the table above as 'pyloric'. In other cases, of course, the ulcer is pyloric, not by a process of elimination but in point of fact.

The figures in the table are worthy of note. What strikes us most is the large percentage of duodenal as opposed to pyloric or gastric ulcers. We would remark here that the number of perforated ulcers operated upon at the Royal Victoria Infirmary, Newcastle-upon-Tyne, is very large (in 1928, for example, the number was 210; in 1929, 221), and when glancing through any volume of notes one cannot fail to notice the large percentage (usually in the neighbourhood of 90 per cent) of duodenal ulcers in cases of acute perforation. The percentage in this series is 90·7. In view of this it is interesting to note in the literature articles on the subject recording a much smaller percentage of duodenal ulcers. Lewisohn,[32] for example, reports only 18 'duodenal and pyloric' ulcers in 33 cases (55 per cent); Bryce[7] 100 duodenal ulcers in 153 cases (65 per cent); Deaver and Pfeiffer[11] 38 duodenal ulcers in 55 cases (69 per cent); while Gibson,[19] Judine,[29] and Fermaud[18] record the low percentages of 55, 48, and 40 respectively in their series. McCreery[34] reports a preponderance of gastric ulcers over duodenal ulcer in his cases, 15 being gastric and 10 duodenal.

In all our duodenal cases, the ulcer was situated on the anterior or antero-superior surface of the duodenum. Both gastric ulcers were situated on the lesser curvature near the cardiac end. There was no case of multiple perforations, of multiple duodenal or gastric ulcers, or of associated duodenal and gastric ulcers.

While it is the commonly expressed opinion that in cases of acute perforation in females the ulcer is more often gastric than doudenal, in the series we are reporting all six females had perforated duodenal ulcers, while the only two cases of perforated gastric ulcer occurred in males.

Age Incidence.—*Table II* gives the age incidence in our series of cases.

Table II.—Age Incidence in 64 Cases of Peptic Ulcer.

Age	Males	Females	Totals
10–19	2	—	2
20–29	11	2	13
30–39	18	1	19
40–49	14	2	16
50–59	9	—	9
60–69	4	1	5
Total	58	6	64

The oldest male was 67, the oldest female 69; the youngest male was 17, the youngest female 28. The average age of the males was 40 years; of the females 41·5 years.

In more detail it may be stated that both cases of gastric ulcer are included in *Table II*; they were aged 25 and 55 respectively, giving an average age of 40 years. Therefore, had they been considered separately, the average age of the duodenopyloric group would still be 40.

From these figures it is seen that the most prevalent decade of life in which the perforation occurred in this series is the fourth, while the third, fifth, and sixth decades are strongly represented. The number of cases occurring in the more extreme decades is almost negligible, but it proves that acute perforation of a peptic ulcer may occur at almost any age, in regard to which Smith[54] makes the interesting statement that acute perforation has been reported in an infant of 2 months and in a woman aged 77. Harrison[24] recently reported a case of perforated duodenal ulcer in an infant 10 weeks old and gives references to eight authors who have each reported a case in infants under one year. The further bearing of this factor on the remote results of acute perforated ulcer will be considered later.

Brown,[5] Fermaud,[18] and Robitshek[47] each shows in his series an age incidence which runs parallel to that recorded by us.

Length of History.—A consideration of the length of history in these cases is of interest, as it shows that it can be divided into two distinct groups. In 56 cases (88 per cent) a history was obtained of gastric symptoms—usually the typical ulcer syndrome—extending over a period of years, while in the much smaller group of the remaining 8 cases, 4 had a history of stomach trouble for only three or four weeks, and in the other 4 the acute pain of perforation was the initial symptom.

Amongst others who have made note of the presence of a long history in the great majority of cases of acute perforated ulcer are Colp,[9] Henry,[25] Moynihan,[37] Sherren,[52] Dineen,[13] and Robitshek.[47] It seems obvious, then that the length of history has a definite bearing upon the perforation incidence of peptic ulcer, and one cannot but come to the conclusion that the older the ulcer becomes, the more likely it is to perforate.

Treatment.—In this series all the cases were treated by simple suture, the ulcer thereby being infolded, except in one case in which gastro-enterostomy was performed in addition to the suture of the ulcer.

The closure of the perforation was brought about by the following standard procedure. Several interrupted Lembert catgut sutures (the number depending upon the size of the ulcer, but on the average 3 to 5) are passed over the ulcer from side to side parallel to the long axis of the duodenum, bites being taken in healthy tissue as close to the sides of the ulcer as possible. When these are tied, the ulcer is infolded and the perforation closed, the line of closure being at right angles to the longitudinal axis of the abdomen. As an extra precaution against leakage, a continuous Lembert suture of catgut is superimposed to invaginate the line of closure. Small tags of omentum, if readily available, may be brought into use by being stitched over the suture line—this is merely a refinement and not an essential in the closure of the perforation. We do not claim that this type of suture has any particular advantage over the ordinary purse-string (unless it be that it interferes less with the blood-supply to the edges of the sutured ulcer), and there is no doubt that the type of suture used is largely a personal matter. The danger of

immediate stenosis following the infolding of the ulcer by either method is apparently negligible.

After the ulcer has been sutured, the peritoneal cavity is then mopped out and the abdominal wound closed. We do not consider drainage necessary unless there is a large amount of fluid present with much soiling and infection of the viscera, when a suprapubic drain is inserted for twenty-four to forty-eight hours.

Results.—

1. *Immediate Results.—Table III* shows the mortality in the present series of cases.

Table III.—Immediate Mortality in 64 Cases of Peptic Ulcer.

Time After Operation	Males	Deaths	Females	Deaths
Under 12 hours	47	1	4	0
Over 12 hours	11	2	2	0
Totals.—				
Under 12 hours	51 cases with 1 death (0·5 per cent)			
Over 12 hours	13 ,, ,, 2 ,, (15·0 per cent)			
Grand Total.—	64 ,, ,, 3 ,, (4·7 per cent)			

All three deaths were in cases where the ulcer was duodenal, although the gastric cases, generally considered to be more lethal, were operated on ten and fourteen and a half hours respectively after perforation. It will be seen from the table that the mortality affected only the males.

The results shown corroborate a commonly accepted fact—namely, that the longer the time between the moment of perforation and operation, the higher will be the resulting mortality.

2. *Remote Results.*—Of the 61 cases remaining out of the 64 operated upon, we were unable to trace 13 (including both cases of gastric ulcer), leaving 48 of whom we have knowledge. Four have died since the operation, the causes being respectively cardiovascular disease, bronchopneumonia, recurrent perforation with fatal peritonitis five months after the previous operation, and sudden death from an unascertainable cause about five weeks after operation, the patient being up and about and feeling quite well to within a few minutes before his death.

The results of the remaining 44 cases are as follows :—

Satisfactory	17	38·6 per cent	63·6 per cent
Fair..	11	25·0 ,, ,,	
Poor..	16	36·4 ,, ,,	

All these cases were treated by suture alone. The one case treated by suture and primary gastro-enterostomy is unfortunately numbered amongst those whom we have been unable to trace.

A note of explanation with regard to the terms 'satisfactory', 'fair', and 'poor' is necessary :—

By 'satisfactory' is meant symptomatic cure. The word 'cure' in describing the results of these cases is purposely avoided, for one cannot assume that the absence of symptoms means necessarily that the ulcer has healed, for we must acknowledge the existence of the silent ulcer.

By 'fair' is meant return of symptoms in a degree not sufficiently severe

to prevent the patient's going about his work and being a useful member of the community. Many of these patients are so little troubled that they refuse to submit to a further operation.

What is the underlying pathology in these cases? Symptomatology can help us only a little. For instance, 2 of these cases have had a mild attack of hæmatemesis, while 2 others have noticed black stools on one occasion. We think we can reasonably assume that in these cases the ulcer was still active at the time when these symptoms were noted. In 4 cases slight pain only at irregular intervals was complained of. The pathological significance of this is doubtful, for it by no means necessarily indicates the presence of an active ulcer. The same may be said of the occasional vomiting complained of by 3 other cases. A barium meal might help to elucidate the pathology, but in this respect it must be remembered that a deformity of the duodenal bulb can be caused by a healed ulcer as well as one which is active.

By 'poor' is meant a return of symptoms severe enough to incapacitate the patient. In some cases the symptoms are those referable to an active ulcer, in others they are indicative of the onset of the complication of pyloric stenosis.

Interpreting the results, we see that in this series a little over a third have had no further trouble at all, a quarter can get along well enough to refuse further operation, and a little over a third have received no lasting benefit at all from operation.

In the paragraph on the 'length of history' we made mention of 8 cases, 4 of whom had a history of stomach trouble for only three or four weeks, while in the other 4 the acute pain of perforation was the initial symptom, and stated that the bearing of this factor on the end-results would be considered later. It was of great interest to us to be able to trace 6 of these cases and to find that in all the result is satisfactory.

Perhaps even more interesting is the fact that in 7 of these 8 cases the pathology found was that of a chronic ulcer—that is, induration around the perforation—proving that an ulcer may be present some time before it gives rise to either symptoms or complications.

Secondary Operations.—Judging by the literature, the majority of surgeons find that after simple suture a certain percentage of cases develop symptoms suggestive either of a recurrent ulcer or of complications, and of sufficient severity to necessitate further operation. The percentage of such cases in the present series is 36·4—a little over a third. This figure agrees with that of Pool and Dineen.[46] Brenner,[4] Smith,[54] and Southam[62] have each reported a smaller percentage of these cases. Stewart and Barber[56] are exceptional in that they have not found it necessary to perform a single secondary operation.

On the 16 cases (36·4 per cent) whose results have been classified as poor, 10 have had a second operation performed. In 8 of the 10 gastro-enterostomy was done and the results are as follows:—

Satisfactory	3	(37·5 per cent)
Fair	2	(25·0 per cent)
Poor	3	(37·5 per cent)

The results run parallel with those obtained after simple suture—a little

over a third have had no further trouble, a quarter of the number improved, and a little over a third have received no benefit.

Recurrence of Symptoms after Operation.—*Table IV* shows the times of recurrence of symptoms in the 27 cases comprising the fair and poor results.

Table IV.—Post-operative Recurrence of Symptoms in Peptic Ulcer.

Months after Operation	Number
0–6	7
7–12	5
13–18	6
19–24	3
Over two years	6

These figures show that in over 50 per cent of these cases the onset of the recurrence of symptoms did not take place until over a year had elapsed after their operation, and in nearly 25 per cent the onset was delayed to over two years—in 2 cases three and a half and five years respectively. Hence we must disagree with Bryce,[7] who holds the view that any return of symptoms after simple closure of a perforation will take place within two years. Furthermore, the figures in the above table in themselves sound a warning note in regard to the too early publication of results claiming 'cures' after treatment of perforated ulcer.

Review of the Surgical Procedures Adopted in Cases of Acute Perforated Ulcer.—Perhaps it is not out of place to discuss the various types of operative procedures which are used in the treatment of acute perforated ulcer and to give a brief historical outline of their development. They may be summarized under four headings: (1) Suture alone; (2) Suture followed by (primary) gastro-enterostomy; (3) Excision of the ulcer and suture (pyloroplasty); (4) Partial gastroduodenal resection.

We have read that Roux[48] in 1883 performed the first operation for perforated ulcer, that Mikulicz[35] is given the credit for the first (unsuccessful) suture of a perforated ulcer in 1889, and that Kriege,[31] in 1892, was the first to suture such an ulcer with success. For several years this simple procedure remained unassailed as the standard method of treatment, until Braun[3] in 1897 introduced the performance of gastro-enterostomy in addition to suture of the ulcer. Moynihan[36] in 1901 urged that a gastro-enterostomy should be performed at the time of closure in order to counteract the pyloric stenosis which sometimes followed the operation of infolding of the ulcer by suture. Deaver[10] has proved himself the great champion of this operation in the U.S.A. Ever since its inception, the performance of this further additional surgical procedure has been, and still is, the subject of a great deal of controversy.

It is of interest that Guthrie,[22] who sent out a questionnaire to numerous American Surgeons with regard to the advisability of performing primary gastro-enterostomy, says it is obvious that by far the greater number are conservative and employ simple suture only.

Suture of the ulcer alone or combined with a primary gastro-enterostomy are both only palliative measures, and soon the idea of a radical treatment bore fruit. It was introduced first in 1909 by Dowden,[15] who excised the ulcer in the longitudinal axis of the duodenum and sutured the opening, thus formed, in the transverse axis. This operation had the double advantage of not only removing the ulcer but also of actually widening the lumen of a hollow viscus at a point where stenosis reasonably might have been expected.

Bolder spirits in time conceived the idea of extending the area to be removed so as to include the whole of the ulcer-bearing area of the stomach and duodenum. Haberer,[23] in 1919, was apparently the first to attempt to popularize resection in cases of perforated ulcers, and was followed soon afterwards by Eunike,[17] Massari,[33] and Schwarzmann,[51] who all reported further successful cases, so that partial gastrectomy and duodenectomy as the treatment for acute perforated ulcer is of quite recent development. Its practice has been confined, so far as we can ascertain, almost entirely to Continental clinics, and is not indulged in to any extent by British or American surgeons. Not every Continental surgeon, however, is in favour of resection, for both Brunner[6] and Noetzel[40] condemn the procedure as unnecessary and dangerous.

Each of the four methods of treatment has its advocates and its opponents, and since this is so, it is obvious that there is still a wide divergence of opinion on the subject of the treatment of acute perforated peptic ulcer. Perhaps it would be well, at this point, to examine the claims put forward by the advocates of each method in proof of its superiority over the others.

Everyone realizes that the acute perforation of a peptic ulcer is a catastrophe of such magnitude as to threaten seriously the life of the afflicted patient. The hypothesis that it is sufficient to ward off the immediate danger without taking thought of the future forms the main consideration upon which is based the claim that *simple suture* of the ulcer is the treatment indicated. The patient in his precarious state is thereby subjected to the least possible amount of operative interference, and the prognosis, so far as life is concerned, is improved. In other words, any operative procedure other than suture is not only unnecessary, but, by prolonging the operation and tending to spread infection in the peritoneal cavity, endangers the life of the patient. Further, immediate stenosis following the suture of an ulcer is, to all intents and purposes, unknown to occur, and cases in which it develops at a later date can then be dealt with accordingly. In this respect, one surgeon has remarked that it is better to have a living patient with pyloric stenosis than a dead one without. Hæmorrhage and recurrence of perforation are also spoken of as being exceptional sequelæ after simple suture. It has been claimed that a perforated ulcer, having been sutured, then undergoes cure in nearly all cases —one of the strongest arguments—if it were true—in favour of suture as the best treatment.

There arises next the advisability of performing a (primary) *gastro-enterostomy* at the time of suture. The arguments against this—as against *any* procedure further than suture—have been indicated above. However, those who are in favour of this form of treatment point out that, in the first

place, it does not add to the mortality in early cases in spite of the prolongation of the operation and of the accusation that infection will be spread, but may actually be instrumental in reducing it. Secondly, they argue that even if no immediate stenosis develops after simple suture, at all events gastro-enterostomy relieves the tension on the suture infolding the ulcer and at a later date guards against hæmorrhage, recurrence of perforation, and the effects of a subsequent stenosis which may occur. Thirdly, they say that the anastomosis is beneficial by permitting of increased alkalinization of the gastric juice. Fourthly, that suture alone does *not* cure the ulcer in the high percentage of cases claimed by its advocates. Fifthly, that the incidence of gastrojejunal ulcer, the bugbear of gastro-enterostomy, is very low.

The reasons given by Dowden[15] for his introduction of *excision* of the perforated ulcer were twofold. In the first place, the ulcer is got rid of entirely or at least to a great extent, and, secondly, the healthier tissue exposed is much more suitable for rapid and safe suturing. Moreover, by performing a plastic operation when suturing up the opening made by the excision of the ulcer, the lumen of the gut is actually increased in a place where following simple suture a stenosis might reasonably be expected to occur at a later date. The disadvantages are obvious and are mentioned by Dowden himself. Only specially selected cases can be dealt with in this manner, others are too unapproachable and at the best difficult to close; any fixity of the ulcer by adhesions to the posterior abdominal wall also tends to make the operation very lengthy and difficult, if not impossible.

The main reasons for the adoption of *partial gastrectomy* as the treatment for perforated ulcer are: first, the success which has attended the treatment of chronic ulcers by this method; secondly, dissatisfaction with the results obtained by the palliative or less radical procedures of suture alone, suture with primary gastro-enterostomy, and local excision followed by pyloroplasty; thirdly, that removal of the ulcer-bearing area definitely saves the patient from such complications as hæmorrhage, recurrence of perforation, or pyloric stenosis. The performance of such a lengthy and severe operation on a patient acutely ill from such a condition as a perforated ulcer, when a much simpler one is all that is necessary to save the patient's life, is strongly opposed by many surgeons who uphold the view that the first principle of emergency surgery is to ward off the immediate danger, without considering the future.

Comparison of Results—Early and Late.—On the whole the mortality percentage of the present series is eminently satisfactory, especially when compared with that of other series of cases. Statistics, of course, are unsatisfactory, for, after all, each case is an individual with a susceptibility and power of resistance to disease peculiar to himself and not admitting of comparison with that of his fellow-sufferer. However, they are the only means we possess in our endeavours to assess the value of any particular operative treatment.

In an article of this length we can give the mortality percentages of only a limited number of series of cases after treatment by the different methods. In considering figures representing total mortality, series with a larger number of late cases will naturally have higher percentages—another factor which tends to decrease in value statistics appertaining to the condition under consideration.

IMMEDIATE RESULTS OBTAINED BY OTHER AUTHORS AFTER THE VARIOUS METHODS OF TREATMENT.—

1. *Simple Suture.*—The following list shows the total mortality percentage in series of cases treated by simple suture of the ulcer, and are taken from recent articles on the subject: Brown[5] 39, Deaver and Pfeiffer[11] 17, Dunbar[16] 29, Gibson[19] 18·6, Morrison[38] 47, Moynihan[37] (duodenal cases) 38, Platou[45] 20, Smith[54] 12, Southam[62] 9, Stenbuck[55] 38·3, Stewart and Barber[56] 8·3, Turner[57] 14·6, Urrutria[58] 15·7.

From this it would appear that an average mortality following this method of treatment is 20 to 30 per cent. Amongst these authors the few who published tables showing the relationship of mortality to the time of operation after perforation all prove by their figures that the earlier the operative treatment is undertaken the less the mortality. For instance, although Southam's total mortality was 9 per cent, 34 of his cases operated on under twenty-four hours all recovered.

2. *Suture and Primary Gastro-enterostomy.*—The figures representing mortality percentages in cases treated by suture and primary gastro-enterostomy present some interesting contrasts to those shown in the last paragraph. Brown,[5] Deaver and Pfeiffer,[11] Morrison,[38] Moynihan,[37] Platou,[45] and Stenbuck[55] all report a smaller percentage of mortalities, this being most marked in the cases of Deaver and Pfeiffer (5·5 per cent by this method compared with 17 per cent of the suture alone), and Morrison (3 per cent—47 per cent after suture alone), while Platou, whose mortality after simple suture was 20 per cent, had no deaths by this method. On the other hand, Urrutria showed an increase in his mortality-rate by over 4 per cent, and Judine[29] and Walton[59] had respective rates of 38·8 and 35·2 per cent.

3. *Excision of Ulcer and Pyloroplasty.*—The reported series of cases treated by excision and pyloroplasty consist of small numbers only, the largest being that of Moynihan,[37] these being 22 cases with 1 death, a mortality of 4·5 per cent. Dowden[15] and Grimault[21] had no deaths. Hinton[26] had a mortality of 8·3 per cent, while that of Williams and Walsh[60] was no less than 33 per cent. Bager,[1] of Stockholm, did 78 cases with a mortality of 11·5 per cent, and lost none of 45 cases operated on within six hours after perforation.

4. *Partial Duodenectomy and Partial Gastrectomy.*—The results shown by men from Continental clinics who prefer radical measures in the treatment of acute perforated ulcer demand the closest consideration, especially in view of the fact that at the present time the great majority of surgeons show a distinct preference for conservative surgery and are inclined to condemn resection unreservedly.

Judine,[29] of Moscow, tells us that he has sought to do resection in all cases except those in which: (1) Intoxication is advanced; (2) The patients are too old; and (3) Resection is difficult. As a result of his activities he has performed 47 resections with 6 deaths, a mortality of only 12·7 per cent; moreover, in 2 of these cases total, not partial, gastrectomy was done, and both patients survived. He attributes his success largely to the use of splanchnic anæsthesia. Pierre Duval, in his discussion of Judine's results,

comments favourably on 'this magnificent series', considering it an unanswerable argument in favour of this resection treatment for perforated ulcer which he had tried to establish in a thesis three years previously.

Doberauer,[14] of Carlsbad, records 11 resections for perforated ulcer with 3 deaths (27 per cent mortality). Odelberg,[41] of Stockholm, has performed 20 partial gastrectomies for perforated ulcer with only 1 death (5 per cent mortality). His only death occurred in a case which had been perforated seventeen hours, and he attributes the fatality to having seen the patient first when it was too late. Mulleder and Neuberger[39] reported 21 resections with 1 death (4·7 per cent mortality). Hromada and Newman[28] report 19 resections with 14 recoveries (26 per cent mortality). They state that all these cases showed widespread peritonitis, and in all more than two-thirds of the stomach was removed. Schwarz[50] reports a series of 10 cases; 9 of these were operated on under twelve hours after perforation and survived. His only death occurred in a patient who had been perforated for eighteen hours.

Every surgeon must be impressed by the figures quoted above, since in some series the mortality is less than that resulting from the treatment of the perforated ulcer by the simplest possible method—namely, laparotomy and suture. It is obvious, however, that such results can only be obtained where the operator has more than average ability and experience in gastric surgery, quite apart from the fact that the patient's general condition must be good.

The sphere of partial gastrectomy as the treatment for perforated ulcer must necessarily be always restricted to clinics specializing in gastric surgery. We are quite sure that it would be unwise for the average general surgeon to attempt to establish this large resection operation as his routine treatment for perforated ulcer, and think he would be well advised to content himself with some less formidable procedure. We think our experience of a 0·5 per cent mortality in cases operated upon under twelve hours, with a total mortality of 4·7 per cent, fully justifies the simplest operative measures—that is, suture alone.

Remote Results Obtained by Other Authors after the Various Methods of Treatment.—

1. *Simple Suture.*—Much has been made of the claim that suture of a perforated ulcer leads to its subsequent cure. Stewart and Barber,[56] for example, found that all of their 22 cases 'during the past five years' which had recovered from operation (suture alone) were well enough not to acknowledge that they were sufficiently uncomfortable to allow themselves to be re-operated on. Gibson[19] states that " In general, patients who recover from a perforation are cured of the ulcer. The great majority are restored to good health and are free from stomach symptoms ": and he adds that these patients do so well that he has been led to refrain from doing probably unnecessary operations as a routine. He followed up 93 patients, the results being 'excellent' in 41 and 'satisfactory' in 31.

In Southam's[62] series, 28 out of 37 patients have remained free from symptoms 'up to periods of two years' after simple suture, and 5 have slight symptoms only. Smith reports that in only 4 cases out of 35 which recovered from the operation was he asked for further advice some months after discharge. Pool and Dineen[46] report 60 per cent of patients well of those who

have recovered from operation (suture alone). Some of these had been operated on twelve years previous to publication.

Platou[45] reports results after suture not nearly so good as those above. Of 25 cases, 3 were re-operated on for perforation within a year, 8 have had gastro-enterostomy done since because of return of symptoms, and 8 are complaining enough to be constantly under medical supervision, so that only some 25 per cent showed anything like a satisfactory result. Pannett[43] believes that the true percentage of cures following simple suture is about 50.

Although we do not agree with those who state that simple suture of a perforated ulcer will lead to its ultimate cure in every, or nearly every, case, yet our personal experience leads us to believe that 60 to 70 per cent of patients treated in this way will have little or no further gastric symptoms. Hence we hold the view expressed by Pool and Dineen,[46] who consider that a definitely indicated later gastro-enterostomy is preferable to subjecting all cases to this procedure as a prophylactic measure, thus saving many patients unnecessary subjection to the possible dangers and discomforts of the operation.

2. *Suture and Primary Gastro-enterostomy.*—From our own series we cannot claim any familiarity with personal results following the performance of suture and primary gastro-enterostomy for perforated ulcer, but it is of interest to examine, for purposes of comparison, those of other writers.

Collinson[8] found that of 27 cases treated in this manner, 70 per cent are well, while of 26 cases treated by suture alone 65 per cent are well, most of the remainder having had gastro-enterostomy with satisfactory results. Deaver and Pfeiffer[11] report satisfactory results in all of 17 cases subjected to primary gastro-enterostomy, and are of the opinion that accumulated statistics and more accurate follow-up records are dispelling the fallacy that a successful operation for perforation cures ulcer, and tend to show that primary gastro-enterostomy lessens the likelihood of future ulcer symptoms and complications. Deaver,[10] we know, has long been the great American advocate of primary gastro-enterostomy in cases of perforated ulcer. In his latest writing he confirms his opinion, though for the past two years he has been making a wide removal of the pyloric sphincter muscle. Dineen[13] obtained 70 per cent good results with suture and primary gastro-enterostomy, as against 80 per cent good results following suture alone.

Lewisohn,[32] who has had experience of both methods, found that closure of an acute perforation, either with or without gastro-enterostomy, failed to cure in 39 per cent of cases. Fermaud[18] claims 90 per cent cures with suture and gastro-enterostomy and 50 per cent cures with suture alone. Platou[45] has done 15 cases in this manner with 100 per cent good results. Urrutria,[58] in 12 cases, has had to perform secondary operations for gastrojejunal ulcer in no fewer than 4—truly a high percentage. He also quotes more than twenty authors who have reported cases of gastrojejunal ulceration following gastro-enterostomy for acute perforating ulcer, and believes that the danger of secondary ulceration is a real one. Williams and Walsh[60] report that in both cases in which primary gastro-enterostomy was done there was a recurrence of perforation at the same site.

3. *Excision of Ulcer and Pyloroplasty.*—The operation of excision of the ulcer, when situated in the duodenopyloric region, followed by a plastic

operation, never seems to have gained much popularity since its introduction by Dowden. It has, however, some staunch enthusiasts.

Dunbar[16] says that the operation has yielded splendid results and does not take more than two minutes longer than simple suture. He also states that no case in which this operation was done has returned complaining of stomach symptoms, and that several of these cases have been X-rayed, when no delay has been shown in the stomach.

Grimault[21] has done 10 cases over a period of ten years, and all of them are well. He says the operation is indicated in late cases where suture or infolding is difficult. Although Williams and Walsh[60] reported a large mortality in cases treated by this method, yet in 5 out of their 6 surviving cases the result has been satisfactory. Other authors who speak well of the operation, and show encouraging results, are Dehelly,[12] Girode,[20] and Basset.[2] On the other hand, Hinton[26] has followed up 9 cases and found 22 per cent well. In view of these results he is of the opinion that one is not justified in continuing to use this type of operation. Rowlands and Turner[49] quote 12 cases treated by this method, of which 5 have had recurrence of symptoms.

4. *Partial Resections.*—Those who have published late results after partial gastrectomy are very enthusiastic about them. Hromada and Newman[28] have followed up 13 of their 14 surviving cases for one to two years, and find that all are free from gastric distress, have good appetites, and have put on weight. Furthermore, all have returned to their previous occupations. Their experience thus leads them to consider resection, even in the presence of peritonitis, the best treatment, and that older methods should be used only when no other method is available. Paul,[44] from von Haberer's Clinic, considers that follow-up reports show this to be a more satisfactory operation than less radical measures. Odelberg[41] has traced 8 of 10 cases who have been operated upon over a year. He found that all were capable of work, that 7 were symptom-free, and that the remaining case had periodic pain.

CONCLUSIONS.

1. A series of 64 cases of acute perforated peptic ulcer is reported: 63 were treated by simple suture and 1 by suture plus gastro-enterostomy. The mortality-rate in cases operated on under twelve hours was 0·5 per cent, and in cases over twelve hours, 15·0 per cent. The total mortality was 4·7 per cent. We are of the opinion that these low figures fully justify the use of simple suture as the routine treatment of acute perforated ulcer.

2. The remote results of simple suture, suture plus gastro-enterostomy, and excision and pyloroplasty are not so good as those recorded after resection. On this account we feel that in treating recurrent ulcers or complications, one should be prepared to consider partial resection rather than palliative measures as a secondary operation.

3. We found that approximately one-third of our cases eventually had to have a secondary operation performed, the remaining two-thirds being in such good health as to preclude the necessity for further operative interference.

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CARCINOMA OF THE CÆCUM.

A DISCUSSION OF ITS INCIDENCE, DIAGNOSIS, AND TREATMENT, WITH A REPORT OF TWENTY-FIVE PERSONAL CASES.

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If the rectum is excluded, 10 per cent of cases of cancer of the large intestine occur in the cæcum. During the period 1920–9 inclusive, 322 cases of carcinoma of the large intestine, other than those occurring in the rectum, were admitted to King's College Hospital; of these 31 were cases of carcinoma of the cæcum. It might be thought that the percentage of cæcal cancers would be higher, considering the irritation to which the cæcum must be subject owing to the fluid contents of the small intestine being squirted through the ileo-cæcal valve and impinging upon its outer wall. On the other hand, the contents of the cæcum are normally fluid, and thus when once the cæcum is full, its mucosa is not nearly so liable to irritation as is that of the distal (left) part of the colon, where the contents are more solid, owing to the absorption of water which takes place chiefly in the proximal (right) part of the colon.

Development of the Cæcum.—About the ninth week of intra-uterine life, the abdominal viscera begin to recede within the parietes, the fœtal peritoneal cavity being at this stage sufficiently large to contain all the abdominal viscera. The return of the umbilical loop of the intestine is accompanied by an important process of rotation of the intestinal tube around the axis of the superior mesenteric artery. The small intestine enters the peritoneal cavity below and to the left of the large intestine and its mesocolon. The upper part of the large intestine together with its mesocolon come into contact with the posterior wall of the omental bursa with which they fuse, and thus the transverse mesocolon is formed. The cæcum is, at an early stage, immediately below and in contact with the liver. At this stage the cæcum may be regarded as marking the right extremity of the transverse colon. The liver and cæcum eventually become separated one from the other. The separation is effected by three growth factors: (1) A relative (not actual) diminution in size of the liver—the liver may be regarded as shrinking headwards; (2) A relatively rapid elongation of the trunk; (3) An actual displacement caudalwards of the cæcum into the right iliac fossa—this displacement is of relatively small extent. As the liver and the cæcum become displaced, one from the other, the large gut distal to the cæcum elongates, and thus the ascending colon makes its appearance. Under certain but somewhat rare circumstances, the cæcum undergoes no relative or actual displacement from its original position,

but persists in contact with the liver and follows this viscus in its displacement headwards. In such cases no ascending colon exists. In other cases the actual migration downwards of the cæcum into the right iliac fossa fails to take place, and the cæcum occupies a higher position than is usually the case, and is found at some little distance above the iliac fossa and leads into a relatively short ascending colon. Sometimes rotation of the gut fails to occur. In such cases the colon occupies the left lower portion of the abdominal cavity, has no connection with the great omentum, and the cæcum is found on the left side of the abdomen. A very rare condition exists in which, owing to the adhesion of its mesentery to the posterior wall of the omental bursa in early fœtal life, the cæcum actually lies in the transverse mesocolon.

Such developmental abnormalities, the majority of which are fortunately rare, must be kept in mind, as carcinoma of the cæcum might be mistaken for carcinoma of some part of the ascending, transverse, or sigmoid colon, for which entirely different surgical treatment would be requisite.

Surgical Anatomy of the Cæcum.—The main points of importance are its: (1) Attachments; (2) Blood-supply; (3) Lymphatic drainage.

Attachments.—The cæcum is, as a rule, completely invested by peritoneum, and for this reason is a mobile viscus. Occasionally the postero-superior wall has no peritoneal investment, and is applied to the posterior abdominal wall to which it is fastened by areolar tissue. Generally speaking, therefore, the surgical mobilization of the cæcum may be said to be free from difficulty.

Blood-supply.—The blood-supply of the cæcum is derived from the artery to the mid-gut—namely, the superior mesenteric artery. The vessels concerned are the anterior and posterior cæcal arteries, branches of the ileocolic artery, a terminal derivative of the superior mesenteric. The supplying vessels are more easily seen if the cæcum is examined from behind. The posterior cæcal branch is the larger of the two, and gives off the artery to the appendix. It can be easily realized that if an attempt is made to ablate the cæcum alone, some of the collateral vessels are endangered. These are more particularly the ileal branch of the ileocolic artery, which supplies the terminal 8 in. of the ileum; and the right colic artery, which is responsible for the vascularization of the ascending colon and part of the transverse colon.

In order to avoid the risk of gangrene, a resection for carcinoma of the cæcum must include not only the cæcum itself but also the terminal 6 or 8 in. of the ileum, the ascending colon, and the proximal 3 or 4 in. of the transverse colon. In actual practice an anastomosis is usually first established between the terminal ileum and the middle of the transverse colon. Ten days later the resection is effected.

Lymphatic System.—The lymphatic vessels of the cæcum correspond fairly accurately to the vascular channels. The lymphatic glands may be conveniently grouped as follows: (1) A local group comprising: (*a*) Anterior cæcal glands; (*b*) Posterior cæcal glands. (2) An outlying system comprising: (*a*) Appendicular glands; (*b*) Ileal glands; (*c*) Right colic glands. The arrangement of these glands can be seen on examining *Fig.* 30. The outlying glands drain into the superior mesenteric group of the pre-aortic lymphatic glands. Any or all of these lymphatic glands may be involved in cases of carcinoma of the cæcum.

It has been estimated by Craig and MacCarty that the lymph nodes are invaded in 32 per cent of cases. This we consider a very generous computation, when it is remembered that the usual enlargement met with is due to a purely inflammatory reaction which subsides when the absorbing area is removed. Communications between the lymphatic vessels of the appendix and cæcum and those of the broad ligament and iliac lymphatic vessels have been described. More recent observations, however, show that there is, at any rate, no primary connection. Nevertheless, extension of carcinoma may take place along the subserous spaces, and secondarily involve the pelvic organs and lymphatic glands.

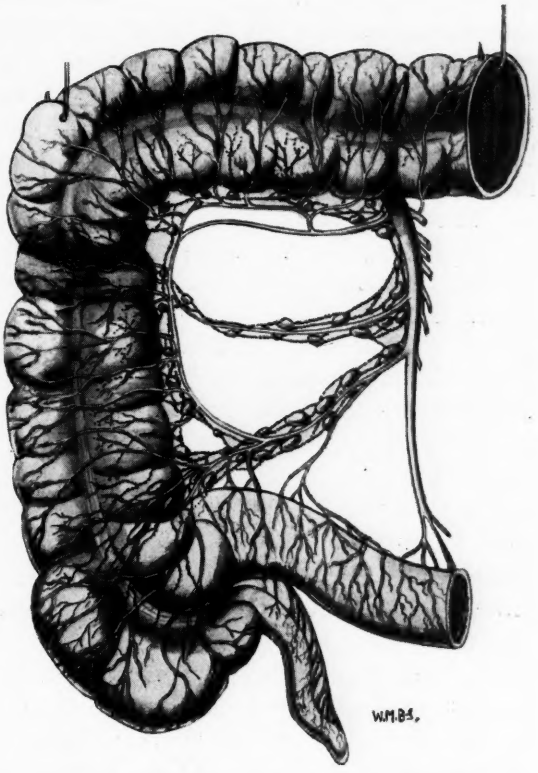

FIG. 30.—Drawing showing the lymphatic drainage of the cæcum and proximal portion of the colon.

Etiology of Carcinoma of the Cæcum.—The youngest case of carcinoma of the cæcum is one reported by Chajutin, which occurred in a girl aged 14 years; the majority of cases, however, occur from the fifth decade onwards. In 100 cases collected from the Mayo Clinic by Craig and MacCarty the average age incidence was 49, the disease being twice as common in males as in females. In our own personal series there were 19 male and 6 female cases—a higher proportion of males than is shown by the American figures. With regard to the habit of the individual, constipation can hardly be looked upon as a factor in its causation, otherwise the condition would be more likely to occur in females than in males.

It is a significant fact that carcinoma occurs commonly in those parts of the alimentary canal which are developed from the 'fore' and 'hind' gut. The small intestine, cæcum, ascending colon, and hepatic flexure, which are derived from the 'mid' gut, are far less commonly affected with the disease. Carcinoma in the small intestine is very rare, and accounts for 2 per cent or less of cases of cancer of the whole intestinal tract. The reaction of the contents of the stomach and the large bowel is acid, while that of the small intestine is alkaline. In the former cancer is common; in the latter it is very rare. Some trauma, which may be regarded as a biochemical trauma, must affect the mucosa lining the outer wall of the cæcum as the alkaline juices of the small intestine are squirted through the ileocæcal valve and impinge upon it.

Can the problem of malignant disease be elucidated in terms of hydrogen-ion concentration? Does not the painting of the skin of rats with tar or a constant application of crude acids favour the appearance of epithelioma? Again, is not the application of radium a means of altering the reaction of the tissue juices *in vivo* with the disappearance of the neoplasm in some cases? In the present state of our knowledge it is impossible to state the cause of the disease, but what better field could there be for speculative thought?

Pathology.—Microscopically, cancer of the cæcum conforms to one of several histological types: (1) Adenocarcinoma; (2) Fibrocarcinoma; (3) Colloid carcinoma; (4) Carcinoma arising in a polypus.

1. *Adenocarcinoma* in the cæcum is the commonest type, and does not differ materially from the same type arising in other parts of the intestinal tract. The acini are lined with typical columnar cells which stain deeply. The acini are irregular in size. Infiltration occurs into the submucosa and may extend into the muscle coat, but usually not deeply or widely.

2. *Fibrocarcinoma* undergoes ulceration in most cases. Fibrosis is variable, but may be very marked. The cells are atypical and are seen in small groups, infiltrating the muscular coat deeply. Infiltration is a marked feature of the growths.

3. *Colloid carcinoma*, better termed 'mucoid degeneration'. This occurs in the adenocarcinoma and all structure may be lost. Small groups of cells only may remain. The mucoid material stains with muci-carmine and exhibits a typical appearance.

4. *Carcinoma arising from polypi* are rare in the cæcum, but have been reported. The carcinoma is of the adenocarcinomatous type. Polypi may show a papillary form of growth. If mucoid degeneration has occurred, the diagnosis of malignancy may be very difficult.

The growth commences in the mucous membrane as an adenocarcinoma, and if we consider the outer wall of the cæcum opposite the ileocæcal valve as the commonest site, the growth first extends downwards to the caput cæci and later extends upwards from the caput cæci to the ileocæcal valve. Owing to the method in which the carcinoma spreads, the ileocæcal valve is not involved until a comparatively late date.

The majority of cases of carcinoma of the cæcum come up for treatment owing to the recognition of a lump in the right iliac fossa. In some cases the growth tends to project into the lumen of the cæcum as a large, fungating cauliflower-like mass (*see Fig.* 32), and in such cases septic absorption and toxæmia occur, and a leucocytosis may be present. In other cases, however, the growth infiltrates the wall, and causes, in some cases, a carcinomatosis of the peritoneum; ultimately the liver may be involved in a carcinomatous capsule (*see Fig.* 39). Colloid degeneration may occur in any type of carcinoma of the cæcum, and is probably as common as in the stomach. Metastatic deposits in the liver from carcinoma of the cæcum may show the typical multiple, yet somewhat isolated, hard areas with umbilication which occur on the surface of the liver (*see Fig.* 35). On the other hand, the liver may be so infiltrated with secondary deposits that no normal liver-tissue can be seen with the naked eye. Again, the liver may be surrounded by a carcinomatous capsule and yet have no actual deposits within it (*see Fig.* 39).

Microscopically, carcinoma of the cæcum is a typical adenocarcinoma (*see Figs.* 34, 37), and frequently the inflammatory reaction due to the superficial septic condition of the tumour can be easily seen.

Symptomatology.—The symptoms are very variable and cannot be said to conform to any definite type. Perhaps the most constant feature is the presence of a palpable tumour. This, we are convinced, is due to the fact that the usual origin of the growth is on the outer wall of the cæcum, whence it extends downwards into the caput cæci, and thence upwards along the inner wall until the ileocæcal valve is more or less occluded. When this happens, symptoms of chronic obstruction arise. There is rarely any pain except in very advanced cases, but constipation is a common symptom. Some degree of anæmia is usually present. Intestinal obstruction is uncommon, except in very advanced cases where the ileocæcal valve is almost completely occluded. There is commonly tenderness in the right iliac fossa in the region of the palpable tumour, and for this reason the condition is sometimes diagnosed as appendicitis. A leucocytosis is present in about 50 per cent of cases and is undoubtedly due to septic absorption from the tumour in patients with good resistance; the presence of leucocytosis is another factor which favours the diagnosis of appendicitis.

In a third of the recorded cases there is no history whatever of loss of weight. It is a remarkable fact that few cases ever complain of nausea. The average duration of symptoms before operation varies between nine months and two years. In our series the shortest period was three weeks, while the longest was three years. In five cases only was visible peristalsis present. This is a rather interesting fact, as it is stated in students' text-books that carcinoma of the cæcum is frequently attended by visible peristalsis of the terminal coils of the small intestine. Blood was noticed in the stools in three of our cases only.

Differential Diagnosis.—A correct diagnosis of carcinoma of the cæcum should be made in the majority of cases, as so few of them are masked by symptoms due to acute intestinal obstruction.

As a palpable swelling in the right iliac fossa is nearly always present, a differential diagnosis has to be made from ileocæcal tuberculosis, abscess of the appendix, and actinomycosis of the cæcum or appendix. Ileocæcal tuberculosis is a rare disease in the London area, and the age incidence is much lower than that of cancer of the cæcum. Loss of weight, anorexia, and passage of blood and mucus in the stools are marked and significant symptoms. Appendicular abscess is generally more painful and is not so resistant on palpation as a neoplasm of the cæcum. In actinomycosis of the cæcum the diagnosis is rarely made except in those cases in which a sinus or sinuses are present and yellow granules are seen in the pus.

Perhaps the most useful aid to differential diagnosis is an X-ray examination. By means of a barium meal or enema, a correct diagnosis can usually be made. In carcinoma of the cæcum the filling defect of the cæcum can usually be seen (*see Fig.* 36). In appendicular abscess the cæcal outline is usually normal. In ileocæcal tuberculosis the terminal ileum is involved as well as the cæcum and a thin narrow channel can be seen leading from the pelvis into a small contracted cæcum. In actinomycosis of the cæcum, although

the cæcal capacity is reduced by the thickening in its walls, there is no marked irregularity of the outline as is seen in cases of carcinoma of the cæcum.

Treatment.—There can be no doubt that carcinoma of the cæcum can only be treated satisfactorily by surgical means. X-ray and radium therapy are of little use. The surgical treatment demands a two-stage operation. Although excision of the growth and an ileocolostomy have been successfully performed at one operation in a few recorded cases, the correct procedure should be first to effect a lateral anastomosis between the terminal ileum and the middle of the transverse colon, and after an interval of a week or ten days, to excise the last 6 or 8 in. of the ileum, the cæcum, ascending colon, and hepatic flexure. There is often some difficulty in peritonizing the raw surface which is left on the posterior abdominal wall in the region of the hepatic flexure; this raw surface, however, can easily be covered over with the omentum, which can be fixed there by a few interrupted sutures.

In one case only in our series was excision of the growth together with an ileocolostomy performed at one and the same operation. The patient died seven days later (*Case* 16). In cases complicated by intestinal obstruction, ileocolostomy should be performed, and the anastomosis should be reinforced by an omental graft to obviate the possibility of leakage through the suture holes in the distended small intestine.

CASE REPORTS.

Case 1.—Mr. A. J., age 58, was admitted to King's College Hospital, on July 11, 1915, complaining of slight pain in the abdomen, which his doctor thought was due to appendicitis. The patient had complained of slight nausea for some time, but had vomited on only two occasions, the day prior to his admission to hospital. There was no history of constipation, and the man had always enjoyed good health until three weeks before coming to hospital.

On Examination.—The patient was found to be a well-built and muscular man, with a furred tongue and a temperature of 90°. The abdomen was distended, there was marked tenderness in the right iliac fossa, and a definite lump could be easily felt in that situation. A leucocyte count revealed 22,000 leucocytes per c.mm. A diagnosis of appendix abscess was made, and a laparotomy was done six hours after admission.

Operation.—When the patient was well under the anæsthetic it was found that the lump in the right iliac fossa was freely movable and not fixed. For this reason a paramedian incision was made and the rectus muscle displaced outwards. A large cæcal tumour was seen, an anastomosis between the ileum and the transverse colon was performed, and the wound closed. Twelve days later the growth was excised, including the terminal 6 in. of the ileum, the ascending colon, and the hepatic flexure. The patient made an uninterrupted recovery, and left hospital on Aug. 10. He reported himself as quite fit and well in December, 1930, which is more than fifteen years since his operation.

The specimen (*Fig.* 31) shows a large nodular growth which appears to have originated on the outer wall of the cæcum and grown downwards towards its base. There was no involvement of the ileocæcal valve. Histologically the growth was a typical adenocarcinoma.

Case 2.—Mrs. F. W., age 65, was admitted to hospital on Sept. 18, 1915, with acute intestinal obstruction of three days' duration. Her husband stated that she had suffered from severe constipation on and off for four years and had lost a considerable amount of weight during the last year or so. Her bowels had not acted for six days previous to admission to hospital.

ON EXAMINATION.—Her condition was poor, the abdomen was greatly distended with generalized tenderness. No tumour could be felt and the rectum was empty. An enema was returned clear.

OPERATION.—Under light ether anæsthesia a paramedian incision was made, and a large carcinoma of the cæcum was found. There was considerable ascites, and secondary deposits in the liver were felt. The ileum was quickly anastomosed to the transverse colon, and the abdomen closed. The patient died eighteen hours after the operation.

AUTOPSY.—The growth was found to involve the whole cæcum and had occluded the ileocæcal valve. There was extensive glandular involvement in the ileocæcal angle and along the posterior abdominal wall. The liver was studded with secondary deposits. The growth was a typical adenocarcinoma.

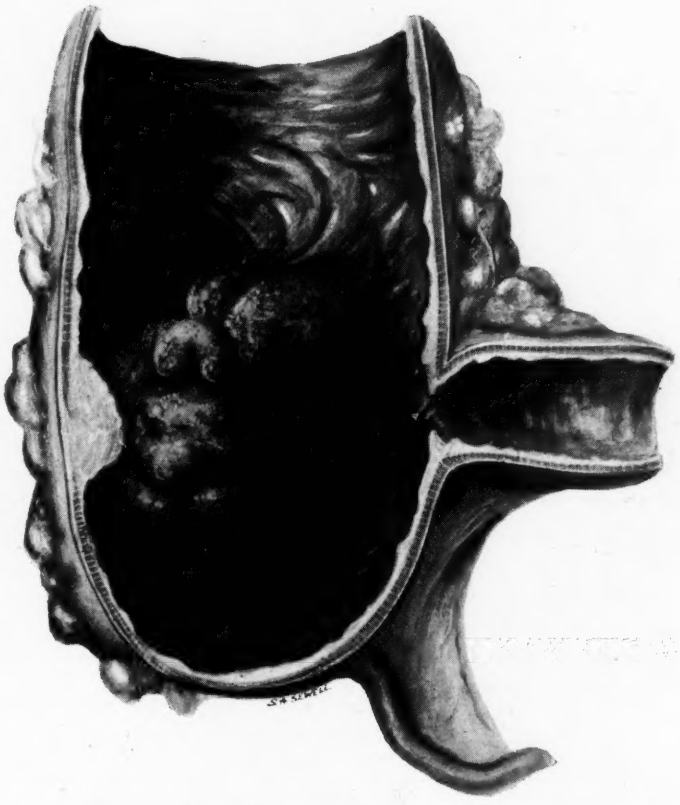

FIG. 31.—*Case* 1. Carcinoma of the cæcum involving the outer wall. The growth exerts no effect on the ileocæcal valve.

Case 3.—Mr. J. A. T., age 62, was admitted to hospital on Dec. 11, 1915, complaining of pain in the abdomen which was intermittent in character, and was first noticed about six months previously.

ON EXAMINATION.—A hard tumour could be easily palpated in the right iliac fossa. There was no abdominal distension, and only slight pain on palpation. The pulse-rate and temperature were normal. The leucocyte count was 8200 per c.mm. An enema produced a good result. The day following admission an operation was performed.

OPERATION.—A right paramedian incision was used to explore the abdomen. A hard growth about the size of a bantam's egg was felt in the cæcum. The rest of the abdomen appeared to be normal, and there was no evidence of any glandular

involvement. The terminal ileum was anastomosed to the middle of the transverse colon, and the abdomen closed. Thirteen days later the growth together with the terminal 6 in. of ileum, the ascending colon, and hepatic flexure were removed. The patient made an uninterrupted recovery. The growth was nodular and somewhat circumscribed, and histologically it was an adenocarcinoma.

We ascertained that this patient died in 1927 from pneumonia. There was no evidence of any recurrence during the twelve years following the operation.

Case 4.—Mr. H. W., age 56, was admitted to hospital on Jan. 11, 1916, complaining of piles.

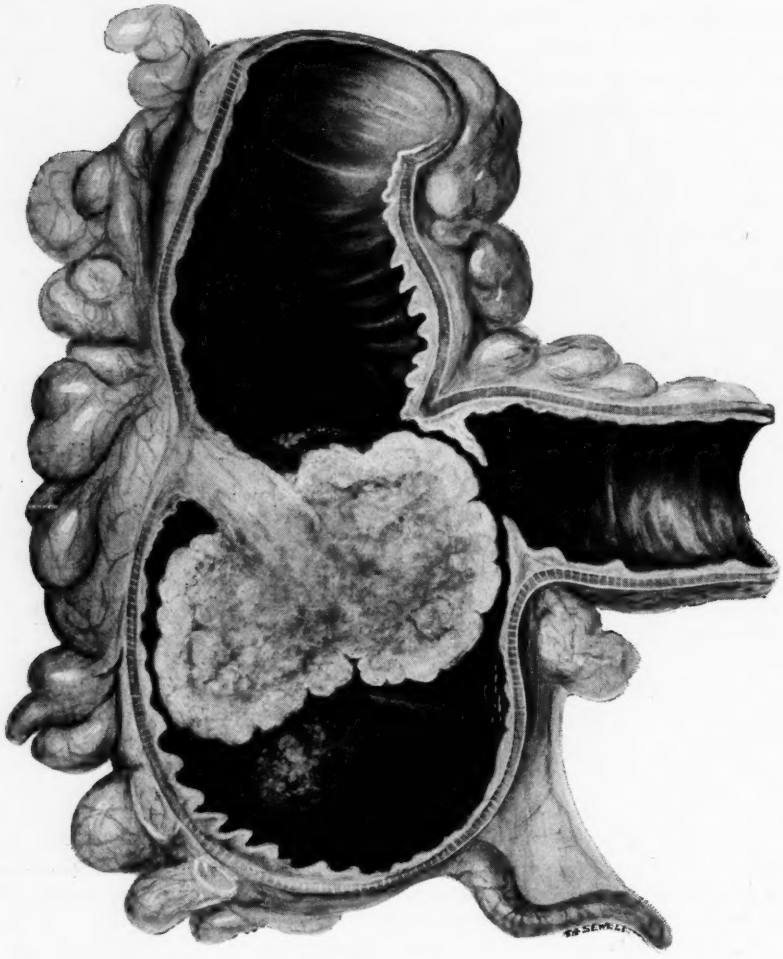

FIG. 32.—*Case* 4. Large polypoid carcinoma of the cæcum. The growth is attached to the outer wall of the cæcum.

ON EXAMINATION.—During a routine examination of the abdomen, a very definite lump was felt in the right iliac fossa. The lump was hard, slightly painful on palpation, but freely movable. On examination of the rectum nothing abnormal was felt or seen, with the exception of three internal piles. A leucocyte count revealed 7600 leucocytes per c.mm. A laparotomy was advised, and on Jan. 15 a right paramedian incision was made, and the abdomen explored. A large growth was felt involving the whole of the cæcum. There was no evidence of glandular involvement or ascites.

OPERATION.—An ileocolostomy was performed. Eleven days later the cæcum, ascending colon, and hepatic flexure were removed. The patient made an excellent recovery.

The growth (*Fig.* 32) was pedunculated, and its pedicle was just opposite the ileocæcal valve. It appeared to fill the whole cæcum, and was just commencing to press on the ileocæcal valve. Histologically it was an adenocarcinoma. The wife of this patient wrote in 1926, saying that her husband had just died from uræmia. He had lived ten years after his operation without any sign of recurrence.

Case 5.—Mr. J. B., age 64, was admitted to hospital on May 5, 1916, as a case of acute appendicitis.

ON EXAMINATION.—The abdomen was distended and painful on palpation. A small hard lump could be felt in the right iliac fossa. The temperature was normal, and the pulse-rate 80. During the twelve hours previous to admission the patient had vomited frequently, and there was a history of melæna. During the last four days there had been no action of the bowels. The patient stated that he had suffered from pains in the abdomen and constipation for the last two years.

OPERATION (May 5).—The abdomen was opened by a right paramedian incision. An extensive carcinoma of the cæcum was exposed. There were numerous enlarged glands along the posterior abdominal wall and a few secondary deposits in the liver. There was a little free fluid in the abdomen. An anastomosis between the terminal ileum and the middle of the transverse colon was performed and the abdomen closed.

The man made an excellent recovery, and was discharged three weeks after the operation. He became jaundiced in August, 1916, and died in September, just four months after the operation. Unfortunately, no histopathology was available in this case.

FIG. 33.—*Case* 6. Moderate-sized cancerous growth commencing on the outer cæcal wall.

Case 6.—Mrs. A. T., age 32, was admitted to hospital on Aug. 4, 1916, as a case of acute appendicitis. She had suffered from intermittent pain in the abdomen for about five months, but during the last two days the pain had become worse, and was localized in the right iliac fossa.

ON EXAMINATION.—The abdomen was not distended. A definite tumour could be felt in the right iliac fossa. There was a leucocytosis of 10,270 per c.mm. The diagnosis of acute appendicitis appeared to be correct.

OPERATION.—A right paramedian incision was made and the abdomen explored; the appendix was quite normal, and was found hanging over the brim of the pelvis.

A hard tumour could be felt in the cæcum about the size of a tangerine orange. The terminal ileum was anastomosed to the middle of the transverse colon, and the abdomen closed. Nine days later the cæcum, ascending colon, and hepatic flexure were removed. The patient made a quick recovery, and left hospital sixteen days after her second operation.

The growth (*Fig.* 33) had started on the outer wall of the cæcum opposite the ileocæcal valve and had extended into the cæcum. There was no obstruction to the ileocæcal valve. Microscopically the growth was an adenocarcinoma.

The patient was alive and well in December, 1930, some fourteen years after the removal of the cancer. This is the youngest patient on whom we have excised a cancer of the cæcum.

Case 7.—Mr. J. T., age 61, came under observation early in 1917 suffering from indigestion and slight loss of weight. A barium meal examination was made on H.M. Hospital Ship *Garth Castle* by Surgeon-Commander Rooney, and a very definite irregularity was seen in the cæcum. The patient gave a history of vague dyspepsia for some two and a half years, but this might have been due to the strain of war-time in the R.N.R.

On Examination.—The patient was found to be a well-built man who appeared to have lost some weight. The examination of the abdomen was negative except for the presence of a hard movable tumour in the right iliac fossa. The leucocyte count was 8164 cells per c.mm.

Operation (April 2, 1917).—Laparotomy was performed under ether anæsthesia, and a large cæcal growth was found. An ileocolostomy was performed between the terminal ileum and the transverse colon. Ten days later the cæcum, ascending colon, and hepatic flexure were excised. There were several glands in the ileocæcal angle; these were removed and on section were found to contain growth.

The patient made a good recovery, but he was invalided out of the service, and died in 1919. He developed an enormous liver and ascites before death.

Case 8.—Mr. W. S., age 58, was admitted to hospital in June, 1918, with a diagnosis of acute appendicitis. He had suffered from slight pain in the abdomen and some constipation for nine months previous to admission.

On Examination.—The patient looked somewhat anæmic and had a furred tongue. The abdomen moved well on respiration; there was slight pain on pressure in the right iliac fossa and a small lump in that situation could be distinguished with difficulty. The temperature was 99° and the pulse-rate 80. Rectal examination simply revealed a loaded rectum. A simple enema produced a large constipated stool. Leucocyte count was 5602 per c.mm. The day following admission the patient had lost practically all his pain, and his temperature was normal. The lump in the right iliac fossa, however, was still present. Operation was advised.

Operation (June 4).—A laparotomy was performed through a right paramedian incision. A growth in the cæcum was discovered, and an ileocolostomy was performed. Ten days later the abdomen was again opened, and the terminal ileum, cæcum, ascending colon, and hepatic flexure were excised. There were no enlarged glands and no secondary growths in the liver. The patient made an uneventful recovery.

The histology of the growth proved it to be an adenocarcinoma. The patient reported himself every six months until 1928, when his wife wrote and said he had died from pneumonia, which caused his death in four days. He had remained quite fit from the time of his operation until his death, some ten years later.

Case 9.—Mr. H. B., age 62, was admitted to hospital on May 6, 1919, with acute intestinal obstruction. He gave a history of abdominal pain for the last two years and marked loss of weight. Constipation had troubled him for years, but for the four days previous to admission he had not been able to get his bowels open even with castor oil. He was vomiting continuously.

On Admission.—The patient was in a collapsed state, with a much distended abdomen. An enema only produced a little flatus. Although the abdomen was

greatly distended, a tumour could be palpated in the right iliac fossa. A blood-count revealed a definite anæmia, the leucocytes being 7460 per c.mm. The patient was kept warm in bed for several hours and his stomach was washed out.

Operation.—This was performed six hours after admission. A paramedian incision was used, and the abdomen opened. There was a considerable amount of ascites; a large cæcal carcinoma was exposed; it was fixed and inoperable. The terminal ileum was anastomosed to the middle of the transverse colon. Secondary deposits were felt in the liver. The patient had a prolonged convalescence, and left hospital six weeks after his operation. After being at home for two months, however, he was re-admitted to hospital because of ascites and some pain in the spine. He died on Aug. 10, 1919, just five months after his operation.

Autopsy.—This revealed generalized carcinomatosis, secondary deposits in the liver, lungs, and vertebral column. The growth was an adenocarcinoma.

Fig. 34.—*Case* 11. Microscopical drawing of the growth removed. (× 65.)

Case 10.—Mr. R. T., age 54, came under observation in January, 1920, complaining of vague pains in the abdomen. He had always enjoyed good health, and worked as a butcher.

On Examination.—Examination in the out-patient department did not reveal anything abnormal. As his pain did not disappear after purgation, a barium-meal examination was made and this revealed definite irregularity in the cæcum. After this it was just possible to make out a cæcal tumour on deep palpation. There was no history of loss of weight or of vomiting or of melæna. A blood-count showed slight anæmia with 5664 leucocytes per c.mm.

Operation (Feb. 2).—The abdomen was opened through a right paramedian incision. A small carcinoma of the cæcum was found, and an ileocolostomy was performed. Ten days later the growth, ascending colon, and hepatic flexure were excised. The patient made an excellent recovery, and returned to work six weeks after his operation. The growth was limited to the outer wall of the cæcum, and was an adenocarcinoma. The patient was alive and well in December, 1930—ten years after his operation.

Case 11.—Mr. G. H., age 58, was sent up to hospital in April, 1920, diagnosed as a case of acute appendicitis. His history was one of vague dyspepsia and abdominal pain for ten months, which had suddenly become worse.

On Admission (April 14).—The temperature was 100° and the pulse 96. There was slight tenderness in the right iliac fossa, and a lump could just be palpated; otherwise the signs were completely negative. The leucocyte count was 6400 cells per c.mm.

Operation.—Five hours after admission operation was performed. Under the anæsthetic the lump in the right iliac fossa could be easily palpated; it was found to be fixed to the cæcum, but freely movable with the cæcum on the posterior abdominal wall. A right paramedian incision was used, and a carcinoma of the cæcum was exposed and brought out of the wound. As the case was an early one and the patient's condition good, we were almost tempted to excise the tumour and do an anastomosis at one and the same time. Better judgement prevailed, however, and an ileocolostomy was performed. Seven days later the growth was excised, together with the terminal ileum, ascending colon, and hepatic flexure. The patient made an uneventful recovery.

The growth had started on the postero-external wall of the cæcum and grown towards the ileocæcal valve, and was just beginning to compress this. The caput cæci was not involved at all. There were no glands in the ileocæcal angle. The tumour was a typical adenocarcinoma (*Fig.* 34). This patient was alive and well in December, 1930—some ten years after the operation.

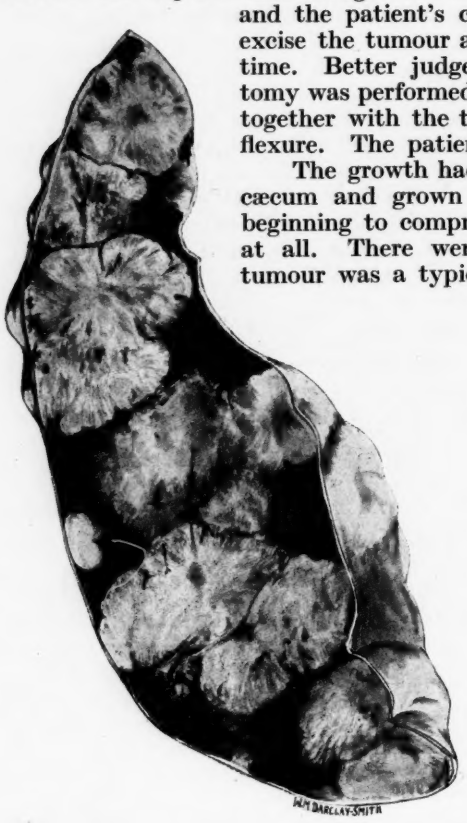

Fig. 35.—*Case* 12. Secondary deposits in the liver. There is more carcinomatous tissue than liver tissue.

Case 12.—Emma Y., age 64, was admitted to hospital on Sept. 19, 1920, with acute intestinal obstruction. Her history was that for the last twenty-one months she had suffered from increasing constipation, culminating in complete cessation of motions six days before admission. The day prior to admission the patient commenced to vomit, and this increased until it became almost constant.

On Examination.—The woman looked extremely ill, with a rapid pulse and a temperature of 97·8°. Her abdomen was greatly distended and tender, and no lumps or tumour could be palpated. Rectal examination simply revealed a ballooned condition of the bowel. An enema was given and returned clear. The stomach was washed out with some sodium bicarbonate solution and a pint of subcutaneous saline was given. The condition of the patient improved somewhat, and six hours after admission operation was decided upon.

Operation.—Under gas and oxygen anæsthesia the abdomen was opened by a right paramedian incision. There was some ascites and secondary deposits in the liver. The primary growth was a large one and situated in the cæcum; it had almost completely occluded the ileocæcal valve. A rapid ileocolostomy was performed, and two pints of 6 per cent gum acacia were given intravenously. The patient recovered from the operation and her bowels acted normally, but the liver became very enlarged and the ascites increased. She died of hepatic insufficiency just two months after her operation.

Autopsy.—There was found to be considerable glandular involvement in the

para-aortic glands. The liver was studded with large umbilicated secondary growths (*Fig.* 35). There were also secondary growths in the lungs and three small growths in the brain. The operation relieved her pain and made her comfortable for the remainder of her existence.

Case 13.—Mr. C. A., age 56, first attended hospital as an out-patient in December, 1920, as his doctor had told him that he was suffering from a gastric ulcer.

On Examination.—Clinical examination did not reveal anything of import except slight tenderness in the right iliac fossa, and the question whether or not this case was one of appendicular gastralgia was raised. A barium meal examination was made, and this showed a normal stomach and duodenum, but a very definite cæcal irregularity (*Fig.* 36). In spite of this examination no definite tumour could be palpated.

Operation.—This was advised on the X-ray findings, and the abdomen was opened by a paramedian incision on Jan. 3, 1921. A small carcinoma of the cæcum was discovered, and an ileocolostomy performed. Ten days later the growth and ascending colon and hepatic flexure were excised. There was no sign of secondary growths in the liver or elsewhere. The patient left the hospital three weeks after his second operation. The growth was a typical adenocarcinoma.

The patient was alive and well in December, 1930, with no sign of any recurrence whatsoever. It has often been stated that much time and material are wasted by patients taking unnecessary barium meals. In this case, however, it was due to the X-ray examination alone that the diagnosis was made at an early stage. A successful operation was performed as the result.

Fig. 36.—*Case* 13. Skiagram showing the filling defect in the cæcum. The amount of barium in the cæcum is very small.

Case 14.—Mr. J. M., age 52, was sent up to hospital on March 4, 1921, with a diagnosis of acute appendicitis. He had suffered from vague abdominal pains and slight constipation for eight months. On the day of admission, as he felt 'off colour' and did not want his food, he visited his panel doctor, who sent him to the hospital.

On Examination.—He did not look very ill; his tongue was furred and his temperature 99°. The abdomen was not distended, but there was slight tenderness in the right iliac fossa, and a lump about the size of a pigeon's egg could be indistinctly palpated. Rectal examination simply revealed a loaded bowel. Leucocyte count showed 5400 cells per c.mm.

Operation.—Under the anæsthetic the tumour in the right iliac fossa became very definite and was found to be freely movable. A right paramedian incision was used to open the abdomen. A small carcinoma of the cæcum was discovered and an ileocolostomy performed. Ten days later the growth, together with the ascending colon and hepatic flexure, was excised. The patient left hospital eighteen days after his second operation; he was alive and well in December, 1930. The only complaint that this patient has, is that his stools are inclined to be loose, and he sometimes has three or four actions a day.

The growth, which was situated on the outer wall of the cæcum, was an adenocarcinoma (*Fig.* 37). There was no evidence of any glandular involvement at the time of the operations.

Case 15.—Mr. T. W., age 58, was admitted to hospital in April, 1922, suffering from chronic obstruction. He had suffered from indigestion for the last eighteen months and this had recently become worse. He now suffered from nausea and occasional vomiting attacks. He had lost weight and was becoming weaker every month.

On Examination.—The man looked definitely ill and anæmic. The abdomen was distended, and a large hard fixed tumour could be felt in the right iliac fossa. Rectal examination was negative. A blood-count revealed a definite anæmia and 6522 leucocytes per c.mm.

Fig. 37.—*Case* 14. Microscopical drawing of the growth removed. (× 65.)

Operation (April 8).—The abdomen was opened by a right paramedian incision and a very large carcinoma of the cæcum exposed. The growth was hard and fixed, and removal was impossible. An ileocolostomy was performed between the terminal ileum and the middle of the transverse colon. There were numerous hard glands on the posterior abdominal wall, but no secondary deposits were seen on the surface of the liver. The patient recovered from his operation very well, and left hospital just over three weeks from the time of his operation. He was re-admitted, however, six months later with ascites and an enormously enlarged liver. He remained in hospital for six weeks and then died.

Autopsy.—There were numerous secondary deposits in the liver and lungs. The primary growth in the cæcum did not appear any larger than it was at the time of the operation, eight months earlier. On microscopical examination the growth proved to be an adenocarcinoma.

Case 16.—Mrs. E. F., age 62, was admitted to hospital on July 2, 1923, suffering from pain in the right iliac fossa and constipation. Two months previously she herself had noticed a lump on the right side of her belly. There was no vomiting and the patient's general condition was good.

On Examination.—A large, hard, movable tumour could easily be palpated in the right iliac fossa. The liver was not enlarged and there was no sign of ascites. A blood examination revealed some anæmia and 7500 leucocytes per c.mm.

Operation (July 3).—The abdomen was opened through a right paramedian incision. A large carcinoma of the cæcum was exposed. The growth appeared to involve the whole cæcum and was beginning to press on the ileocæcal valve. The condition of the patient appeared good and there was no sign of secondary deposits. The growth and last 6 in. of ileum, ascending colon, and hepatic flexure were excised, and the ileum was anastomosed to the transverse colon. The patient suffered from severe shock after the operation, but had her bowels open on the third day and appeared to be going on well. On the seventh day after operation, however, her heart became dilated and she died.

The growth (*Fig.* 38) involved the whole cæcum, and had invaded the cæcal wall in many places. Histologically it was an adenocarcinoma. This patient would in all probability have lived if a two-stage operation had been carried out.

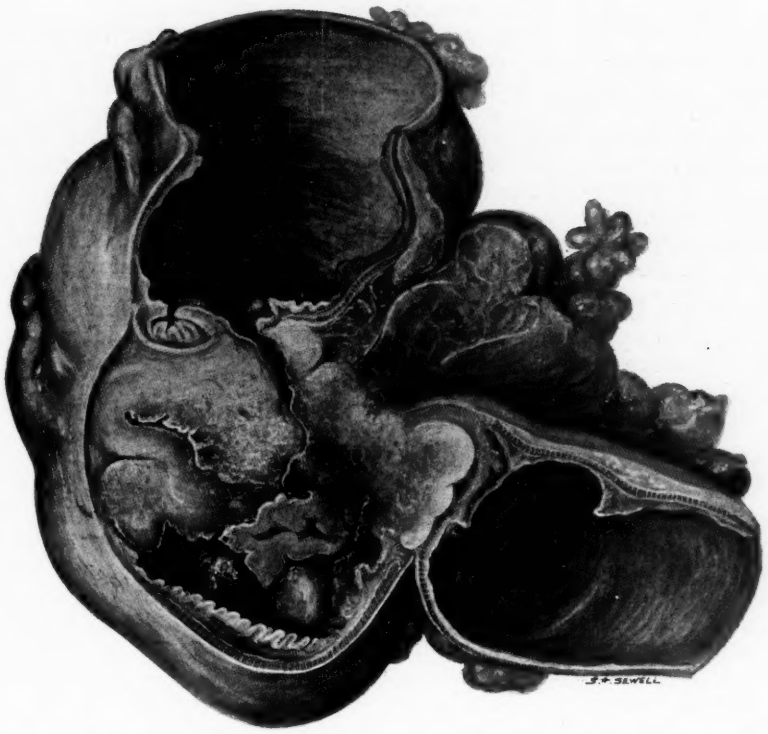

Fig. 38.—*Case* 16. Carcinoma involving the whole cæcum and the ileocæcal valve.

Case 17.—Mr. J. T., age 68, was admitted to hospital in November, 1923, his doctor having noticed a lump in his abdomen. He had complained of pain in the abdomen for about a year, and had suffered from increasing constipation. He stated that he had become weaker during the last few months and had lost weight.

On Examination.—The abdomen was slightly distended, and a large, hard tumour could be felt in the right iliac fossa. The liver was slightly enlarged, but there were no signs of ascites.

Operation (Nov. 12).—The abdomen was opened through a right paramedian incision. A large, fixed carcinoma of the cæcum was exposed with some secondary deposits in the liver. Owing to the fact that the ileocæcal valve appeared to be partially blocked by growth, it was thought necessary to do a short-circuiting operation. The terminal ileum was anastomosed to the transverse colon. The patient recovered from the operation, but did not regain his strength. A month after the

operation there was definite enlargement of the liver and ascites. The patient gradually became weaker and died just two months after his operation.

AUTOPSY.—The liver was found to be three times its normal size and riddled with growth. There were also secondary growths in the lungs. The peritoneum and intestines were studded with secondary nodules. The growth, histologically, was an adenocarcinoma.

Case 18.—Mr. F. K., age 56, came under observation in March, 1924, suffering from hæmorrhoids, and as these did not disappear with the application of lotions and ointments, he was admitted for operation.

ON EXAMINATION.—On routine examination prior to operation a lump was palpated in the right iliac fossa. A leucocyte count was taken, and revealed 7480 cells per c.mm. With the exception of three internal piles the rectal examination was negative. The lump in the iliac fossa did not disappear after purgation, it underwent no change, and was freely movable. The condition was diagnosed as carcinoma of the cæcum, and operation was advised.

OPERATION (April 2).—Laparotomy was performed through a right paramedian incision. A carcinoma of the cæcum was exposed, and an ileocolostomy between the terminal ileum and the transverse colon was performed. Ten days later the growth and the right colon were excised. There was no evidence of any secondary deposits. The patient was able to go home eighteen days after his second operation. The growth was about the size of a tangerine and was limited to the outer wall of the cæcum opposite the ileocæcal valve. There were no enlarged glands in the ileocæcal angle.

Histologically the growth was a typical adenocarcinoma. The patient was alive and well in December, 1930—over seven years after the removal of the growth.

Case 19.—Mr. E. T., age 61, was admitted to hospital on June 4, 1925, with a diagnosis of acute appendicitis. He stated that his bowels had been troublesome for the last eighteen months.

ON EXAMINATION.—His temperature was 90°, pulse 80, respiration rate 20, and the tongue furred. Abdomen moved well on respiration. There was some tenderness in the right iliac fossa and a definite lump about the size of a duck's egg could be palpated. Leucocyte count was 8000 per c.mm. Rectal examination was negative.

OPERATION.—This was performed six hours after admission. Under the anæsthetic the lump in the right iliac fossa was found to be movable. A right paramedian incision was used to open the abdomen. A carcinoma of the cæcum was exposed, and an ileocolostomy performed. On June 14 a second operation was performed, and the growth and the right half of the colon were excised. There was no evidence of any secondary growths. The tumour involved the outer wall and head of the cæcum; the glands in the ileocæcal angle were enlarged, but did not contain any growth. The patient made an uneventful recovery. The growth was an adenocarcinoma.

The patient was alive and well in December, 1930—more than five years after the operation.

Case 20.—This case was reported by Dr. Anson, a former house surgeon to one of us (C. P. G. W.) in the *Lancet* (1927, i, 704).

A man of 65 was admitted to hospital on Aug. 18, 1926, with acute intestinal obstruction. The acute symptoms were relieved on admission by an asafœtida enema. The patient gave a history of a dull pain in the hypogastrium for three weeks. He had also been getting constipated, the motions becoming smaller in quantity and very hard, though of normal shape and colour. There had been no diarrhœa and no blood or mucus in the stools. Defæcation had become increasingly difficult, with much tenesmus. Micturition was normal, except that the quantity of urine had diminished slightly three days after the onset of constipation.

ON EXAMINATION.—The pulse-rate was 84, the temperature 98·4°. The abdomen was distended and prominent; the movements were normal, and there was no rigidity and no tenderness. The hypogastrium was dull to percussion, with shifting

dullness in both flanks. The liver, spleen, and kidneys were not palpable. On rectal examination a hard nodular mass about the size of a large egg was felt in the anterior wall of the rectum 2 in. above the external sphincter. It was not movable, and the surface felt as if it was ulcerated, but no bleeding occurred on examination. A diagnosis of carcinoma of the rectum was made, and as obstructive symptoms increased, a colostomy was performed on Aug. 20.

Operation.—A paramedian incision 3 in. long was made through the left rectus muscle, and the peritoneum was opened. A condition of diffuse carcinomatosis was found, with small nodules over the small gut and colon, and also in the mesentery. About three pints of dark-brown ascitic fluid was drawn off, and a colostomy performed. The gut was opened, a Paul's tube was tied in, the wound was then sutured and packed round with iodoform gauze and a powder of boric and salicylic acids to prevent ascitic oozing.

On the day after the operation the patient vomited 30 oz. of foul coffee-grounds vomit. There was a slight action of the colostomy; although purgatives were given freely, an action of the colostomy became increasingly difficult. The liver and spleen became palpable on Oct. 4, and the patient died on Oct. 23.

Autopsy.—At autopsy the body was greatly emaciated; both lungs showed collapse and bronchopneumonia at the bases. Heart muscle showed brown atrophy. The peritoneal cavity was almost obliterated by adhesions and carcinomatous growth; there were numerous small nodules on the abdominal wall and over the surface of the gut. A huge mass of carcinoma was found beneath the left lobe of the liver, slightly adherent to it and to the stomach, but not invading the stomach wall. On examining the mass felt per rectum, it was found to be a secondary deposit situated between the rectum and bladder. As it was obviously not the primary growth, the viscera were removed *in toto* with part of the posterior abdominal wall; they were hardened in 10 per cent formalin, and a detailed dissection was subsequently carried out.

The dissection showed a large, soft, fungating mass of carcinoma of the typical cauliflower variety filling the cæcum and occluding the ileocæcal valve. The large mass of growth already referred to as lying below the left lobe of the liver was found to be a huge secondary deposit in the pre-aortic glands. Spreading out from it were numerous nodules in the mesentery which were almost confluent at its base, but became more aparsely scattered as the free margin was approached. The splenic hilum was invaded by growth spreading from the region of the cœliac group of pre-aortic glands; the periphery of the liver was invaded by a mass of growth which completely encapsuled the organ, the enclosed liver tissue being perfectly normal (*Fig.* 39). The duodenum and stomach were normal, although part of the greater curvature was adherent to the spleen and to the mass of growth springing from the pre-aortic glands. The pancreas, kidneys, and prostate were normal.

Microscopic examination of the primary growth in the cæcum showed a typical adenocarcinoma with very well-marked colloid degeneration. The secondary deposits in the peritoneum over the small gut and colon and invading the periphery of the liver were of similar structure, as was also the mass lying between the rectum and bladder. This deposit of growth had penetrated the muscular coats of the rectum and invaded the mucosa and was obviously just about to ulcerate in the lumen of the rectum.

This case offers an interesting example of mis-diagnosis, the error being chiefly due to the unusual position of part of the secondary deposit—namely, between the rectum and the bladder. In the light of the above findings it appears that doubts might have been raised as to the correctness of the diagnosis by the continuation of some degree of obstruction after colostomy, and also by the evidence of extensive secondary deposits, although the primary seat of growth in the rectum had not yet ulcerated through into the lumen of the bowel. On the other hand, in the carcinoma of the cæcum secondary deposits are usually late in appearance, and, moreover, relief of obstruction occurring at the ileocæcal valve would not be expected to follow the operation of colostomy.

There seems little doubt that in this case the original attack of acute obstruction relieved by colostomy was due to the combined action of the growth obstructing

the ileocæcal valve, and the secondary mass narrowing the lumen of the rectum below. The failure of the colostomy to act freely and the subsequent increasing obstruction were due to the primary seat of growth obliterating the ileocæcal valve more and more completely, until finally almost complete obstruction again resulted. Another unusual feature is the fact that there was a comparatively early and typical primary growth in the cæcum and very extensive secondary deposits all over the peritoneal cavity.

The case presents rather a striking example of the spread of the carcinoma by at least two well-defined and separate paths—namely, peritoneal and lymphatic spread. The peritoneal spread is shown by the presence of small nodules in the peritoneum of both the anterior and posterior abdominal walls, while the secondary

FIG. 39.—*Case* 20. Secondary carcinomatous capsule surrounding the liver. There were no growths in the substance of the liver.

nodules situated on the surface of the small gut were also seen in the peritoneum. Microscopic examination showed that only the outer portion of the muscular coats of the gut had been invaded, the mucosa being quite intact. A peritoneal spread, moreover, offers the only satisfactory explanation of the rare phenomenon of a complete carcinomatous capsule surrounding the liver and infiltrating its periphery for about an inch, while the central portion was left completely normal with no trace of any secondary deposit in its substance. The mass lying in the rectovesical

pouch which lead to the error in diagnosis was due either to direct peritoneal spread or to a small mass of carcinoma cells becoming detached into the peritoneal cavity and then falling into the rectovesical pouch, there undergoing development. The lymphatic spread is shown by enlargement of the ileocolic groups of glands, which could be traced upwards until the huge secondary mass lying in the superior mesenteric group of pre-aortic glands was reached. Further lymphatic spread from this point along the superior mesenteric afferent lymphatics was shown by the nodules of growth becoming more sparsely scattered through the mesentery as its periphery was approached; while at its base they formed a large confluent mass of carcinoma as already described. The deposit at the splenic hilum was due to the permeation of growth from the superior mesenteric group to the cœliac group of pre-aortic glands and thence along the afferent vessels to the splenic hilum.

It must also be mentioned that two small secondary deposits of growth were found in the sternum, opposite the 7th left and 3rd right ribs respectively. This would appear to indicate some dissemination of the growth by the blood-stream, although no further evidence of such a path of spread could be found.

Case 21.—Mr. R. W., age 54, was admitted to hospital on Feb. 3, 1927, as a case of acute appendicitis. He had suffered from vague abdominal pains on and off for eight months prior to admission.

On Admission.—The temperature was 99·2°, pulse 88, and respirations 20. The abdomen was slightly distended, and a lump could easily be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count was 9500 cells per c.mm.

Operation (Feb. 3).—A right paramedian incision was used to explore the abdomen. A carcinoma of the cæcum was found, and an ileocolostomy performed. Nine days later the growth and right side of the colon were excised. No secondary deposits were seen in the abdomen. The patient left hospital three weeks after his second operation.

Histologically the growth was an adenocarcinoma. The patient was alive and well in December, 1930—nearly four years after his operation.

Case 22.—Mrs. A. B., age 59, was admitted to hospital on Aug. 6, 1927, with acute intestinal obstruction. She had always suffered from constipation, which had become more marked for the last two years. For four days previous to admission the constipation had been absolute and was accompanied by several attacks of vomiting.

On Examination.—The patient was found to be very wasted and anæmic. The abdomen was distended, but in spite of this a tumour could just be felt in the right iliac fossa. Rectal examination was negative. A turpentine enema was returned clear.

Operation.—Laparotomy was performed the same day under gas and oxygen anæsthesia. The mass in the iliac fossa was found to be a large carcinoma of the cæcum involving the ileocæcal valve. The ileum was anastomosed to the transverse colon. Secondary deposits were felt in the liver, and there was free fluid in the peritoneal cavity. The patient recovered from the operation, but the liver increased markedly in size, and she died six weeks after the operation.

Autopsy.—There was generalized carcinomatosis of the peritoneum with multiple secondary growths in the liver. The growth was an adenocarcinoma with marked colloid degeneration.

Case 23.—Mr. J. A., age 55, was admitted to hospital on Jan. 2, 1928, as a case of acute appendicitis. He had suffered from pain in the right iliac fossa on and off for ten months, but on admission the pain had disappeared.

On Examination.—Temperature 100°, pulse 90, respirations 22. There was some tenderness in the right iliac fossa, and on rectal examination no tumour could be felt. Leucocyte count was 6450 cells per c.mm.

Operation.—This was performed through a muscle-split incision; the appendix was inflamed and a small growth was felt in the cæcum. The wound was enlarged

and an ileocolostomy performed. Ten days later the abdomen was opened through a paramedian incision and the cæcum and right half of the colon were excised. The growth was limited to the outer wall of the cæcum, just opposite the ileocæcal valve. There were no secondary deposits inside the abdomen. The patient made an uninterrupted recovery, and was alive and well in December, 1930—just three years after his operation.

Histologically the growth was an adenocarcinoma.

Case 24.—Mr. W. B., age 62, was admitted to hospital, a tumour having been discovered in his abdomen in the out-patient department. He had suffered from constipation for nearly two years, and had been under treatment for this for six months. He had lost a considerable amount of weight during the last year. There was a history of nausea but no actual vomiting

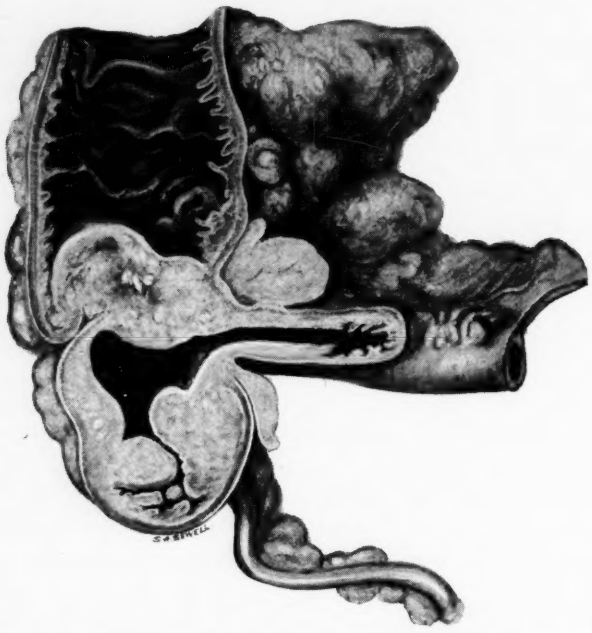

FIG. 40.—*Case* 24. Carcinoma of the cæcum growing into the ileocæcal valve.

ON EXAMINATION.—There was some abdominal distension and a lump could just be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count showed 8000 cells per c.mm.

OPERATION (Feb. 4, 1928).—Laparotomy was performed, and a carcinoma of the cæcum involving the whole cæcum and compressing the ileocæcal valve was exposed. The ileum was anastomosed to the transverse colon. Eleven days later the carcinoma together with the right half of the colon was removed. The patient made an uneventful recovery, and was alive and well in December, 1930—almost three years after the removal of the growth.

The tumour (*Fig.* 40) involved the whole cæcum and was growing into the ileocæcal valve. Histologically it was an adenocarcinoma.

Case 25.—Mrs. H. W., age 69, was admitted to hospital early in January, 1931, complaining of abdominal pain. She had enjoyed good health until six months previous to admission, when she first suffered from nausea. Her bowels had been somewhat costive of late. She was in the habit of taking salts every morning.

ON EXAMINATION.—The patient did not look her age, and seemed quite cheerful. The abdomen was slightly distended, and a hard lump, the size of a cricket ball, could

be palpated in the right iliac fossa. Rectal examination was negative. Leucocyte count showed 11,000 cells per c.mm.

OPERATION (Jan. 7).—The abdomen was opened through a right paramedian incision and a large fixed carcinoma of the cæcum exposed. There were many enlarged hard glands in the ileocæcal angle and on the posterior abdominal wall. An ileocolostomy was performed between the terminal ileum and the transverse colon. The patient made rapid progress, and was discharged from hospital on Jan. 22. When seen six months later the liver was considerably enlarged and there were definite signs of ascites.

SUMMARY.

1. Carcinoma of the cæcum is a disease which is preponderant in the male, and occurs in the fifth and sixth decades; the majority of cases being in the fifties.

2. The primary neoplasm is usually situated opposite the ileocæcal valve. From this fact it may be inferred that trauma is a definite factor and that the trauma is biochemical in nature.

3. The growth is invariably an adenocarcinoma, in which colloid degeneration is quite common.

4. Metastases do not occur until the symptoms have been present for many months; consequently early removal will procure complete immunity.

5. As far as the differential diagnosis is concerned, appendicitis is suspect in the vast majority of cases. In elderly patients the presence of a tumour in the right iliac fossa and absence of vomiting should place the surgeon on his guard. A high leucocyte count is no guide, as septic absorption from the tumour in a patient with good resistance produces leucocytosis.

6. Surgical treatment should consist of: (*a*) An exploratory laparotomy, when ileocolostomy is to be performed should carcinoma be discovered. After an interval of from a week to ten days, this is to be followed by (*b*) Excision of 8 in. of the terminal ileum, cæcum, ascending colon, and hepatic flexure.

7. Where symptoms have been present for any period short of one year, anastomosis and resection offer definite cure. With a few exceptions, when symptoms have lasted more than one year, operation offers no more than one year of life. It may be fairly stated that, after the milestone of one year's symptoms is passed, the duration of the patient's life is in inverse proportion to the subsequent months.

In conclusion we should like to express our thanks to Dr. H. A. Lucas for much help in the preparation and examination of the microscopical structure of the majority of our cases. We wish also to tender our grateful thanks to Miss Mary Barclay-Smith, Mr. Ford, and Mr. Sewell for their excellent illustrations.

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A REPORT ON THE STRANGEWAYS COLLECTION OF RHEUMATOID JOINTS IN THE MUSEUM OF THE ROYAL COLLEGE OF SURGEONS.

By R. LAWFORD KNAGGS,

With Photo-micrographs by G. H. RODMAN.

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PART I.

HISTORY OF THE STRANGEWAYS COLLECTION.

THE Strangeways Collection of Rheumatoid Joints is one result of an effort on the part of a considerable number of medical men and their friends to provide opportunity for the careful and concentrated investigation of particular diseases which might from time to time be selected. The committee included Sir Clifford Albutt, Sir William Church, and Professor Sims Woodhead, and the first subject chosen for study was osteo-arthritis and the chronic rheumatoid conditions. Dr. S. T. P. Strangeways, who had been associated with Professor Kanthack at St. Bartholomew's Hospital, and afterwards at Cambridge, first as a Demonstrator and then as a University Lecturer on Pathology, was entrusted with the management of the inquiry (1905). He received no salary, and, moreover, the committee were indebted to him for much personal effort in collecting funds to finance the small hospital that was started. Cases and specimens were sent to him from many medical men in different parts of the country who were interested in the work, and he was able, by keeping in touch with the physicians in charge of the Poor Law Infirmaries, to obtain a knowledge of many cases, and in due course to acquire many valuable specimens.

In 1908 a special Research Hospital was erected in Hills Road, Cambridge, and here the various researches into the nature of chronic arthritis were carried out by Dr. Strangeways and those willing co-workers who from time to time became associated with him. These researches were published in the Committee's Annual Bulletins.* In the meantime the collection of pathological specimens had been gradually developing into a small museum. The work was interrupted by the War, and the taking over of the Hospital by the military

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* Dr. Strangeways contributed the following articles to the Bulletins of the Committee for the Study of Special Diseases:—

1905. "A Résumé of Some of the More Important Literature on the Etiology and Symptoms of Rheumatoid Arthritis"—with EVA MCCOLL, M.D.
1907. "A Report on Some Points in the Etiology and Onset of 200 Cases of So-called Rheumatoid Arthritis"—with J. BARNES BURT, M.D.
1907. "A Study of Joints from Cases of Rheumatoid Arthritis and Chronic Gout by Means of Skiagrams and Dissection of the Affected Parts".
1907. "An Enquiry into the Value of the Opsonic Index"—with M. P. FITZGERALD and R. J. WHITEMAN.
1907. "A Study of Skiagrams from the Hands of 100 Cases of So-called Rheumatoid Arthritis and Chronic Gout"—with J. BARNES BURT, M.D.
1908. "Conjunctival Reaction to Tuberculin in Arthritic Diseases".
1908. "Report on a Case of Hypertrophic Pulmonary Osteo-arthropathy"—with C. PONDER.
1910. "A Study of Joints from Cases of Rheumatoid Arthritis and Chronic Gout by Means of Skiagrams and Dissections of the Affected Parts"—with EMILY H. MORRIS.
1914. "A Case of So-called Rheumatoid Arthritis due to Pneumococcal Infection".

Proc. Roy. Soc. Med., 1923, xvii, 1.—In a discussion on the etiology and treatment of osteo- and rheumatoid arthritis, Dr. Strangeways made a valuable contribution to the study of rheumatoid arthritis. By the aid of pathological specimens he demonstrated six types of arthritis at present included under that name—viz.: (1) A capsular type; (2) A dry type; (3) The adhesive type; (4) The atrophic form; (5) The villous type; and (6) The infective type. For further details, see the report of the discussion in the *Proceedings*.

authorities. When Dr. Strangeways was able to resume his labours he was gradually led to the study of tissue culture in its bearing upon the growth of articular structures—especially cartilage. The war, therefore, may be considered to have marked the end of the formation of the Strangeways Collection.

Dr. Strangeways died in his sixtieth year, on Dec. 23, 1926, and the Collection was offered by the trustees, Sir Humphry Rolleston, Professor H. R. Dean, and Dr. Malcolm Donaldson, to the Council of the Royal College of Surgeons, by whom it was accepted on July 14, 1927. When the Council of the Royal College of Surgeons took over the Collection, it entrusted to me the task of examining, preparing, and describing the specimens. This report contains the observations I made, and the conclusions I came to, regarding the nature of osteo-arthritis and the chronic rheumatoid conditions during the three years devoted to the investigation of the Collection.

The Collection comprises some 250 specimens of chronic arthritis and a large number of microscopical sections. There is also a typed manuscript in which Dr. Strangeways has described the macro- and microscopical peculiarities met with in his study of diseased joints. The manuscript is illustrated by photographs of specimens and microscopical sections.* These proved of great use in confirming the identity of many of the specimens and of the slides obtained from them.

The majority of the diseased joints were obtained from infirmary inmates, who in many instances were advanced in years and hopelessly crippled. Others were from young people, and many of these had been acquired after surgical excision. Most of the specimens were the subject of advanced disease; many cases were represented by more than one joint, and in several instances a series of joints had been obtained from a single individual.

Clinical notes were supplied with a large number of the specimens; and since the Collection came into possession of the College a good deal of valuable information has been obtained about others. Specimens of osteo-arthritis, rheumatoid arthritis, and gout formed the bulk of the Collection, but there was also an important group of Charcot's joints. Some examples of tuberculous disease and acute septic arthritis had probably been included for the sake of comparison.

THE CORRELATION OF OSTEO-ARTHRITIS, RHEUMATOID ARTHRITIS, AND GOUT.

A careful examination of the different portions of such a comprehensive collection produced a considerable modification of my previous views as to the nature of the rheumatoid affections. At the very outset two difficulties

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* Had Dr. Strangeways lived to complete his work, it is certain that he would have paid a tribute to the co-operation of Mrs. Strangeways—especially in the photographic part of it. It will be seen from the text that the photographs in the manuscript have contributed materially to placing the collection of specimens upon a satisfactory permanent basis; but they also constitute a very useful part of the gift to the College; for the manuscript, which forms three considerable volumes, contains many excellent representations of microscopical preparations which can be consulted when the specimens are being examined. The catalogue description of each specimen illustrated in the manuscript contains a reference to the figures connected with it.

forced themselves to the front: (1) How is osteo-arthritis to be distinguished from rheumatoid arthritis, and what is the difference between them? and (2) What is the position of gout in relation to them?

Before these questions are considered, an epitome of the criteria generally relied upon to discriminate osteo-arthritis from rheumatoid arthritis may be interpolated with advantage.

The salient pathological features of *osteo-arthritis* are:—

1. Vertical fibrillation and splitting of the articular cartilage, which becomes worn away at the sites of pressure and friction.
2. Sclerosis, eburnation, and grooving of the underlying articular bone.
3. Osteophytic outgrowths, more particularly at the articular margins (lipping), and, not infrequently, on the articular surfaces.

Those of *rheumatoid arthritis* are:—

1. Erosion of cartilage independent of friction or pressure, e.g., at the articular periphery.
2. The formation of fibrous adhesions, and of fibrous tissue between the articular surfaces, tending to result in
3. Ankylosis, fibrous or bony.
4. Absence of lipping and other bony changes distinctive of osteo-arthritis—such as eburnation and grooving.
5. Atrophic changes, which are usually pronounced.

The clinical distinction between the two conditions is equally clear-cut. In *osteo-arthritis* there is:—

1. Pain, stiffness, and effusion into the joint, all of which may be but little marked.
2. Grating, or crepitation on movement.
3. Deformity from osteophytic outgrowths.
4. When movement is restricted to the point of fixation, it is due to locking of marginal osteophytes. It is rare to come across any adhesion or ankylosis between articular surfaces, except in certain small joints, in which, normally, movement is of a very limited character.

In *rheumatoid arthritis*:—

1. There is more evidence of inflammation—usually of a subacute character.
2. The swollen joints are apt to assume a fusiform shape owing to the involvement of the peri-articular tissues.
3. There is much pain and limitation of movement, often associated with contractures and muscular atrophy, and occasionally complicated by displacements.
4. Finally, there is a very marked tendency towards rigid ankylosis, which is sometimes bony, but more often fibrous.

THE DIFFERENCE BETWEEN OSTEO- AND RHEUMATOID ARTHRITIS. THE TWO CONDITIONS MERGE INTO EACH OTHER PATHOLOGICALLY AND CLINICALLY.

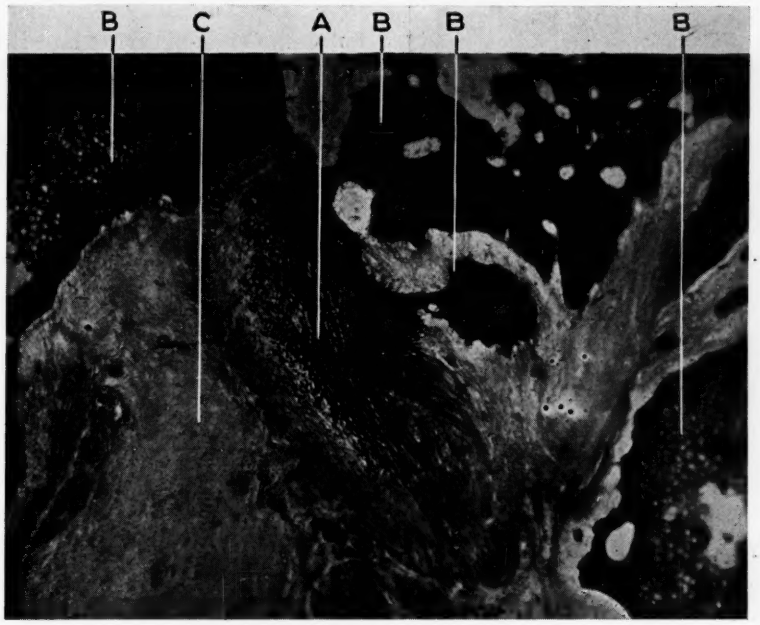
As the investigation proceeded it soon became evident that there is no sharp dividing line between osteo-arthritis and rheumatoid arthritis. It is well known that the two conditions may merge or overlap to some extent, but I was certainly unprepared for the frequency with which, in a collection of unselected cases, some of the signs characteristic of osteo-arthritis were associated with others peculiar to rheumatoid arthritis.

Marginal lipping—looked upon as almost diagnostic of osteo-arthritis—was present in many specimens of unquestionable rheumatoid arthritis: though slight in some, it was so pronounced in others that they might easily

have been mistaken for osteo-arthritic joints, if the evidence in favour of rheumatoid arthritis had not been incontrovertible.

[Lipping arises in different ways. One form results from the collapse of very atrophic cancellous tissue underlying the articular surface, and the squeezing outwards of the marginal portion. This kind of lipping is more likely to be found in rheumatoid arthritis, but even in that affection the usual type, which depends on marginal osteophytes, is the prevailing one.]

Eburnation was present in several rheumatoid arthritic joints. It was not nearly so frequent as lipping, but its occurrence in joints which, to all



FIBROUS DEGENERATION OF BONE.

FIG. 41.—Section from the radius from a Charcot's wrist (left). Emma A., age ?. A, Trabecula undergoing resolution into fibrous tissue. B, Bone degenerating; an early stage of fibrous resolution. C, Space filled with fibrous tissue; hardly any cells present—the consequence of fibrous resolution of bone. (*Specimen* S.C. 93.) (× 50.)

appearances, had been long ankylosed from adhesions and contracted ligaments at once attracted attention. The presence of *grooving* in some instances showed that friction had undoubtedly been present in an earlier stage of the disease.

Ankylosis, on the other hand, which is an end-condition of rheumatoid arthritis, seems specially to affect certain joints in osteo-arthritis. It occurs in the vertebral, carpal, and tarsal articulations, and is doubtless favoured by the slight mobility permitted by the anatomical configuration of the joint surfaces, and by the pressures to which they are subjected. Bony ankylosis is so common in these joints that I had come to look upon it as a natural osteo-arthritic phenomenon.

Ankylosis in rheumatoid arthritis shows no such discrimination. It affects all kinds of joints—both large and small. Intra-articular adhesions,

contracted capsules and ligaments, and muscular spasm and contracture, all play their part, and the bones may be fixed together by bone or fibrous tissue.

The ankylosis in both affections is almost certainly inflammatory in origin, and must not be confused with the 'locking' which occurs occasionally in some of the large joints in osteo-arthritis—notably in the hip. In such cases the articular surfaces are usually sclerosed and eburnated, and, though in contact, are not adherent to one another.

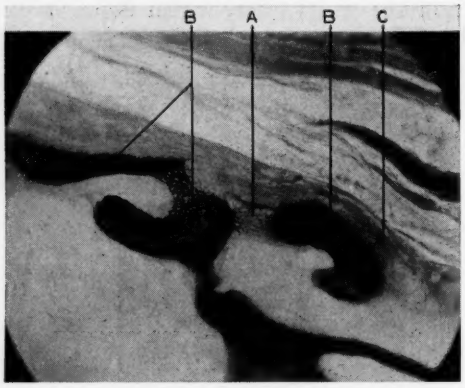


FIG. 42.—Fibrous resolution of bone beneath a fibrous replacement of the articular cartilage. Section from the head of a left humerus. Rheumatoid arthritis. Jessie R., age 70. A, Intermediate portion of a trabecula resolving into fibrous tissue; B, Degenerating trabeculæ; C, Fibrous covering layers, partly resulting from fibrous change in the cartilage, and partly from fibrous degeneration of bone. (*Specimen* S.C. 35—11.) (× 35.)

The Difficulty of Distinguishing between Osteo-arthritic and Rheumatoid Lesions.—The macroscopic phenomena just mentioned do not exhaust the evidence of this tendency of the two forms of arthritis to merge, but they are the most striking examples. Owing to the way in which signs usually considered to be diagnostic of osteo-arthritis strayed into specimens whose general character was that of rheumatoid arthritis, it was often difficult to decide in which category a specimen ought to be placed, and only after consideration of the clinical history, and the obvious inflammatory nature of the affection, were some of them finally labelled 'rheumatoid arthritis'. The difficulty was not a fanciful one. On several occasions expert pathologists first formed the impression that certain specimens were osteo-arthritic, but on more careful examination recognized them as examples of rheumatoid arthritis. To one authority both knees from the same individual were submitted; the disease in one was diagnosed as osteo-arthritis, and in the other as rheumatoid arthritis. Even Dr. Strangeways in his typed manuscript has figured two joints from the same case—one as osteo- and the other as rheumatoid arthritis. (S.C. 47·2, S.C. 47·4. *Figs.* in MSS. 85 and 119.)

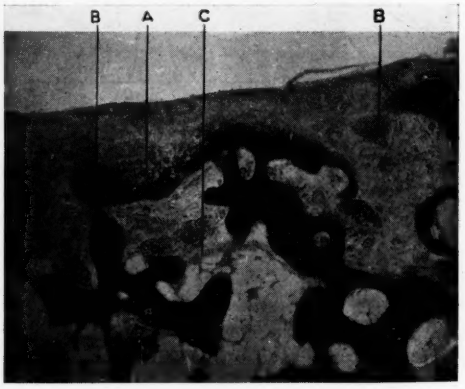


FIG. 43.—Fibrous degeneration of bone. Section from a case of rheumatoid arthritis. Sarah A. H., age 76. Left knee. A, Bone and cartilage degenerating into fibrous tissue; B, Trabeculæ fading into fibrous tissue; C, Strand of fibrous tissue joining two trabeculæ, and representing a resolved portion of bone. (*Specimen* S.C. 41—5.) (× 35.)

Toxic and Other Factors in the Production of the Arthritic Changes.—The bone and joint changes of osteo- and rheumatoid arthritis are thought by some to be the result of a deficiency of some diet factor, and that the two

affections are 'deficiency diseases'. The more general belief is that they are caused by toxins. I am in sympathy with this latter opinion. The toxins may be of micro-organic origin and may obtain entrance to the circulation from some distant focus of infection, or they may be formed locally in an infected joint. They may also enter the circulation by the process of 'sub-infection' described by Adami (*see* Note, p. 160, *Inflammatory and Toxic Diseases of Bone*) in Lane's *Operative Treatment of Chronic Intestinal Obstruction* (4th ed.), and others may be the consequence of disturbed metabolism (Adami, *General Pathology*, i, 342).

Whatever their source or their precise nature, it may be presumed that the toxins circulated to the different joints of the same individual would be identical, and that the phenomena produced would be of similar type, though not necessarily the same in degree. If the type differed, the presence of some other factor would seem probable.

I would suggest that a very natural factor is to be found in the vitality of the joint tissues and their ability to resist or modify the reaction that toxic damage may excite. Most people will agree that variations in these qualities exert an important influence in modifying disease; and a clearer understanding of some of the problems in the pathology of osteo- and rheumatoid arthritis may be reached if it is assumed that such differences *do* affect the joint tissues of those who suffer from these disorders.

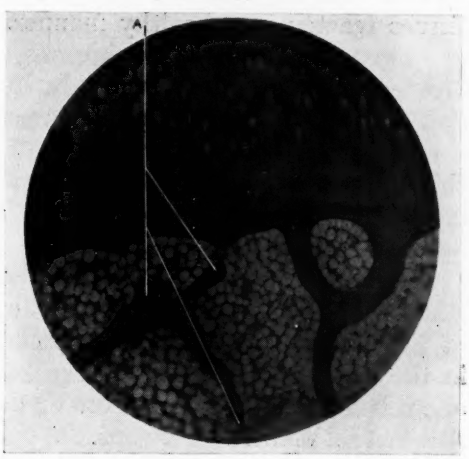


FIG. 44.—Slight signs of fibrous resolution of bone; early signs of cartilage degeneration. Section from a case of rheumatoid arthritis ending in bony ankylosis. Mary G., age 54. From the head of the humerus. The site of this section is more central than those shown in *Figs.* 45 and 46 from the same case, which are near the margin of the articular surface. The cartilage shows proliferation of cells and atrophy of the matrix around. A, Traces of fibrous resolution at the extremities of a trabecula. (*Specimen* S.C. 27—10.)

It is well known that the subjects of osteo-arthritis are usually robust people who have led active and even arduous lives; and that the signs, as a rule, make their appearance in middle age. Rheumatoid arthritis, on the other hand, is more apt to attack the delicate and feeble, whose health has, in many cases, been further impaired by associated circumstances; consequently many young people are included among its victims.

As a probable inference from these facts and theories I have come to look upon the various conditions classified under osteo- and rheumatoid arthritis as representing degrees of the same process, and these degrees as signifying stages in the failing vitality of the articular structures.* The stages start from a state which represents a joint

* Facts would seem to justify an assumption that one joint may be more susceptible to toxic damage than another, or may show an inferior power to react to it, even in the same person. This, indeed, is generally accepted, for it is recognized that a joint may be so

in full health, and descend step by step towards complete disorganization. Thus, on the step below the healthy joint comes osteo-arthritis, and the steps below this correspond to the different varieties of rheumatoid arthritis.

In osteo-arthritis the joint tissues react in a certain way to conditions—presumably of a toxic nature—that injure them, and though not irritated to the point of diapedesis, they are, nevertheless, stimulated to produce bone. In rheumatoid arthritis, on the other hand, the inflammation set up is usually accompanied by leucocytic exudation, and is frequently combined with degenerative changes in capsule, cartilage, and bone—in other words, the inflammatory response to the toxic damage is marked, but the tissues are unable to return to health, and tend to degenerate.

It will be shown that joints take on the rheumatoid arthritic character by reason of inflammatory degeneration of the articular cartilage and bone, and the capsule is also involved. This degeneration is evidence of a diminished tissue vitality; and the frequent presence of osteo-arthritic phenomena, side by side with degenerative changes, points to a period of the disorder when the tissues reacted in a healthy manner.

Final Conclusion.—The investigation of these intermediate cases in the Strangeways Collection has gradually led me to the belief that osteo-arthritis and rheumatoid arthritis are expressions—at opposite ends of the same scale—of a single disease, which I have reason to think is toxic in its origin. On this supposition osteo-arthritis and rheumatoid arthritis are not separate affections, but simply names by which two groups of characteristic pathological changes are distinguished.

When the tissues are healthy and robust the toxic irritation acts as a stimulus to growth processes, and osteo-arthritis results. When the vitality of the tissues is poor, definite inflammatory reaction may be excited, and in the event of the tissues being unable to maintain their vitality in the face of such reaction, degeneration or even disintegration results. In either case rheumatoid arthritis develops.

THE RELATION OF GOUT TO OSTEO- AND RHEUMATOID ARTHRITIS.

Gout is very closely akin to the rheumatoid affections. A urate of soda deposit is the one diagnostic feature of gout, and in its absence it is impossible to distinguish the gouty joint from one that has the characteristics either of osteo-arthritis or of rheumatoid arthritis. Numerous specimens are included in the Collection whose gouty nature is attested by the deposit of urates. Nearly all of them show other arthritic changes, which in many instances completely overshadow the one that justifies their inclusion in the gouty category.

At the present time uric acid and the urates are believed to be a consequence of faulty purin metabolism. They are not thought to be the cause of gout, but to indicate the occurrence of certain metabolic changes, which, in

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impaired by injury, cold, or exposure as to become less able to resist infection by micro-organisms. Moreover, one case will be quoted from which it may be inferred that tissue resistance to toxic influence may vary in different parts of the same joint (*Specimen* S.C. 35–13) (*see Figs.* 49–52).

addition to the uratic deposit, " produce simultaneously other bodies having toxic effects ". (Adami, loc. cit., i, 342.)

It may well be that, while the biurate of soda is being deposited in the joint and the surrounding tissues, the toxic bodies just mentioned set up changes in the articular bone and cartilage which are indistinguishable from those of osteo- or rheumatoid arthritis. It is possible that all the phenomena of gout may be contained within the compass of a single pathological sequence. At the same time it must not be forgotten that almost everyone may become the producer of such toxins as are believed to be instrumental in causing the rheumatoid affections; and therefore the arthritic changes in the gouty joint —apart from the deposition of urate of soda and the inflammation for which it is responsible—may well be due to a twofold toxic attack; one by toxins resulting from disordered purin metabolism, and the other by the same kind of toxins that are supposed to be responsible for the arthritic changes in osteo- and rheumatoid arthritis.

Articular cartilage and other joint tissues seem to have a special attraction for urates, and their deposition is probably the first step in that deterioration which renders a joint susceptible to toxic damage.

FIBROUS AND BONY ANKYLOSIS IN RHEUMATOID ARTHRITIS. THE HISTOLOGICAL CHANGES COMPARED.

The Strangeways Collection contains two series of specimens which I wish to contrast.

Case 1.—The first series (Group S.C. 27) was obtained from a well-nourished woman, aged 54. The disease began in the thumb and then in the great toe when she was 18. It progressed gradually and all the time was attended by great pain. Twelve years before death she could sit up in bed, propped up by pillows. Then she had a bad attack of pain and never sat up again. Her jaw was almost fixed. She could sew and feed herself almost up to the time she died.

Post-mortem.—There was advanced atheroma of the aorta, the kidneys were slightly granular, the muscles fatty, and the spine curved laterally.

Six of the ten specimens from this patient *show bony ankylosis*—namely, both knees, in which all three bones are fused, with subluxation in the left; the left hip, which is dislocated; the left ankle, tarsus, and metatarsus; the right elbow and right wrist. The post-mortem notes state that the left wrist and right ankle were also ankylosed. The bones were very rarefied, and several fractures close to the ankylosed joints were no doubt caused by the tests for mobility at the post-mortem.

Case 2.—The second series (Group S.C. 35) was acquired from a small thin woman, aged 70. The clinical notes tell us only that the limbs were flexed, the joint movements greatly limited, and some of the phalangeal joints ankylosed. One kidney was granular, and pulmonary adhesions, emphysema, and pneumonia of the left base were present.

This series also contains ten specimens—namely, both hips, both knees, and a right ankle with part of the tarsus; also both shoulders (in one the head of the humerus only); both elbows, and the right wrist and hand. The bones entering into the different joints are closely bound together by fibrosed capsules, and the joint cavities are much diminished. Capsular contraction is marked, all the 'slack' in such joints as the shoulder, elbow, hip, knee, and ankle being taken up; for example, in the hip the capsule is attached round the margin of the head of the femur and to the edge of the acetabulum. Quite apart from adhesions, joint movements must have been practically abolished. But though the articular surfaces are thus held in

close contact, and were probably subjected to some degree of pressure under conditions which might be considered favourable to bony fusion, yet *no bony ankylosis is present in any of the large joints.* Indeed, cartilage still covers the whole or the chief part of the articular surfaces, though in places it is atrophied or hypertrophied or transformed into fibrous tissue; and whilst in some instances the joint cavities are seen between the cartilages, in others they are partially occluded by fibrous adhesions. Fusion of two bones occurs only in one of the phalangeal joints, in the carpus, and in one of the astragalo-calcanean joints.

What is the explanation of this striking difference between the two series of specimens? Why is bony ankylosis the rule in the first, and its absence such a noticeable feature of the second?

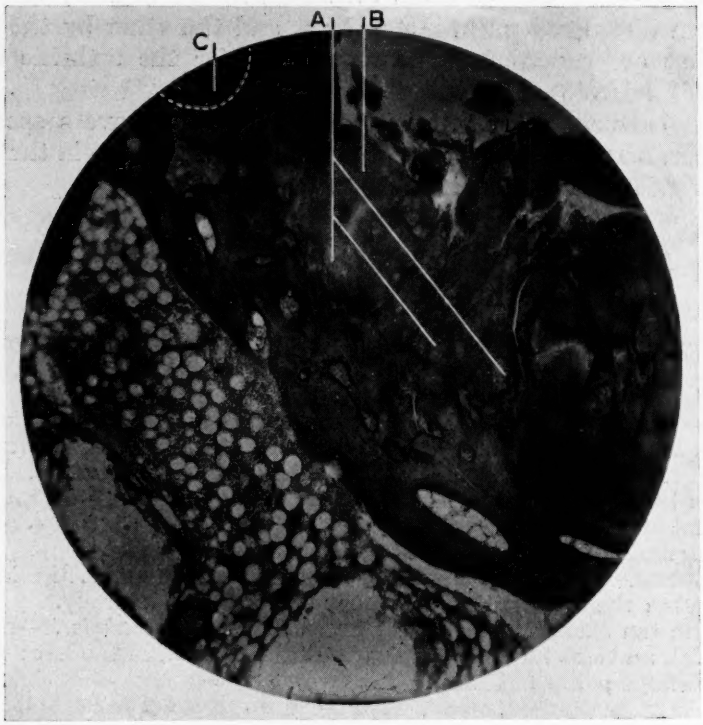

FIG. 45.—Changes in articular cartilage from a case of ankylosing (osseous) rheumatoid arthritis. Section from the head of the same humerus as *Fig.* 44, but nearer the margin. Calcification and ossification are advancing into degenerating cartilage, which is being changed into fibrocartilage on the left, and breaking up into villous processes on the right. Vascular and cellular connective tissue fills many of the spaces in the subchondroid bone and calcified layers, and medullary and fat cells occupy the spaces below. A, Proliferating cells in spaces; B, Fibrosing cartilage: C, Well-marked fibrocartilage. (*Specimen* S.C. 27—10.) (× 90.)

Bony ankylosis, as a rule, begins with the fusion of granulation tissue springing from two opposing articular surfaces denuded of cartilage; often an intrusion of similar tissue from the synovial membrane is incorporated with this interosseous bond. Into this area of fibrosing granulation tissue trabeculæ advance from opposite sides, and coalesce; medullary spaces form, and ultimate fusion of the cancellous tissue and medulla of the separate bones follows. Bone is usually the result of metaplasia of the fibrous tissue.

The mode of union in Case 1 differs from that outlined above. In that case the cartilage assumes the rôle played by the granulation tissue in the typical form.

Microscopical sections were taken from a shoulder and an elbow—from a humerus in each case. In these joints no bony union had so far occurred. The chief changes are in the articular cartilage, and are very similar in both joints. The outstanding feature is the activity of the cartilage cells. The matrix shows a tendency to become fibrous, and to degenerate around the capsules. The cells within the capsules increase rapidly in number, especially

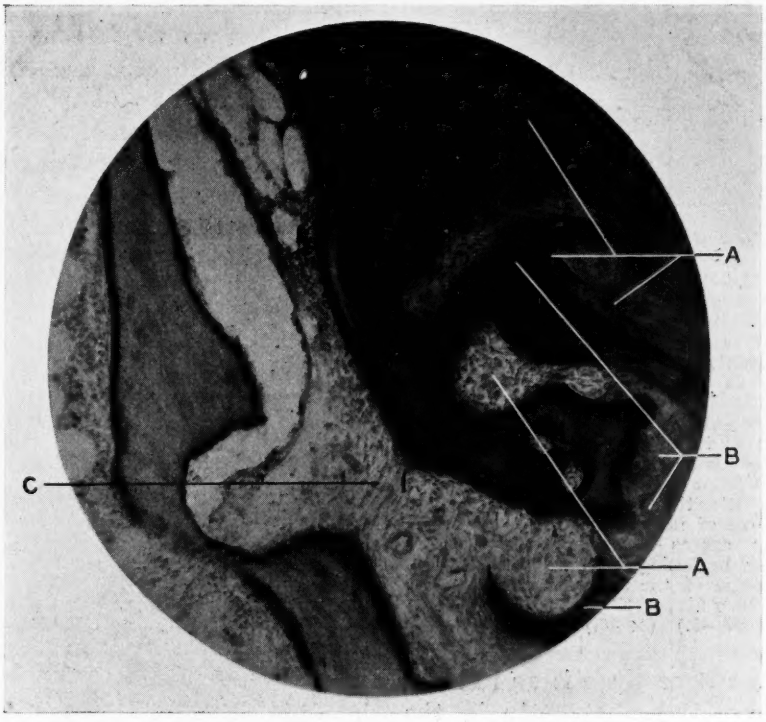

FIG. 46.—Changes in articular cartilage in a case of ankylosing (osseous) rheumatoid arthritis. From the same section as *Fig.* 45, which it joins. The crescent at the right-hand bottom corner can be recognized in *Fig.* 45 (C). A, Fibrocartilage; B, Fibrocartilage metaplasing into bone; C, Very vascular proliferating connective tissue. (*Specimen* S.C. 27—10.) (× 90.)

towards the surface; they maintain a healthy appearance and stain well (*Fig.* 45). A more advanced condition shows the cartilage transformed into a very cellular fibrocartilage in which masses of crowded cartilage cells with tracts of hyaline matrix occur. The calcified zone is less regular, increased in depth, and ill-defined. Ossification is active, bone being produced by metaplasia in calcified cartilage, in fibrocartilage, and in the crowded masses of cartilage cells (*Fig.* 46). In one section a mass of fibrocartilage occupies a medullary space below a much changed portion of the articular cartilage from which it is obviously an outgrowth (*Fig.* 47). The presence of inflammatory cells in the capsule and synovial membrane put the inflammatory nature of the

joint changes beyond doubt.* Associated with these changes there are, in places, others suggestive of commencing degeneration (*Fig.* 48).

From the energetic character of the abnormal ossification going on in the articular cartilage, it may be inferred that bony ankylosis would probably result, if degenerative changes within the cartilage did not progress and prevent it.

The histology of Case 2 is illuminating. Sections from several specimens show that both cartilage and bone behave in a very different fashion to that seen in *Case* 1. In the cartilage the cells are less evenly distributed and tend to disappear, so that the fibrous transformation of the superficial layers shows only sparse elongated nuclei, whereas the deeper layers assume in some parts a fibrocartilaginous, and in others a fibro-connective-tissue character. In the subarticular bone there is an increase in the size and apparently in the number of the bone-cell cavities, and traces of fibrous tissue can often be seen in the substance of the trabeculæ (*Fig.* 42). From the margins of the subchondral bone multitudinous fibres stream outwards and blend with the fibro-cartilage and the fibro-connective-tissue, so that the transition from bone to the altered cartilage is gradual and almost imperceptible (*Fig.* 43).

FIG. 47.—Changes in the articular surface layers in a case of ankylosing (osseous) rheumatoid arthritis. Elbow. Section from lower end of humerus. The parts shown are close to those in *Fig.* 48. A mass of fibrocartilage has formed in a medullary space and degenerative changes are going on in cartilage and bone. A, Degenerative spaces in the subchondroid bone containing fibrocellular connective tissue and giant cells; B, The mass of fibrocartilage in the medullary spaces — ossification is apparently beginning at one point by metaplasia of the fibrous tissue; C, Developing fibrocartilage. (*Specimen* S.C. 27—9.) (× 35.)

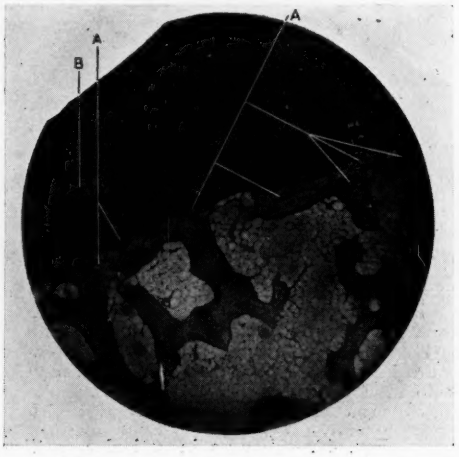

FIG. 48.—Changes in the articular surface structures in a case of ankylosing (osseous) rheumatoid arthritis. Mary G., age 54. Left elbow. Section from lower end of humerus. Calcification and ossification are advancing into degenerating cartilage. The advanced margin of calcification (B) is defined on the left in the middle depth of the cartilage. Above it there is fibrocartilage to the surface, and fibres run into it. A number of small spaces (A) in the subchondroid layer of bone, and two or three involving the cartilage, contain proliferated connective tissue. (*Specimen* S.C. 27—9.)

* The sustained inflammatory reaction going on to diapedesis in *Case* 1 may be compared with the changes usually seen in osteo-arthritis. In that disorder the chief evidence of inflammation is the proliferation of the connective tissue and the stimulation of ossification, but without diapedesis, except of the most trivial character.

Medullary spaces bounded by these degenerating trabeculæ contain a varying amount of fibrous tissue with which the numerous fine fibrils emerging from the bone intermingle (*Fig.* 50). Cells, some probably liberated and altered bone-cells, are numerous in the immediate vicinity of the resolving bone. It is evident that the superficial trabeculæ are slowly disappearing, and being replaced by fibrous tissue. The fibrosing cartilaginous covering is thus being continually recruited on its deep surface, whilst the level of the trabeculæ in contact with it becomes irregular, and tends slowly to recede.

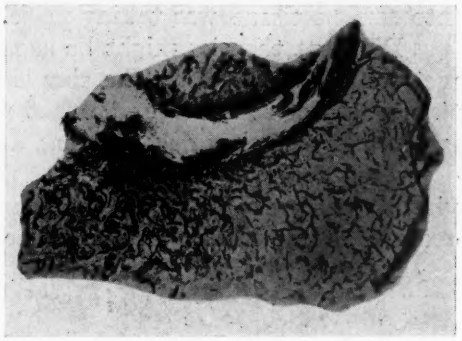

FIG. 49.—Rheumatoid arthritis. Fibrous ankylosis. Left elbow-joint. Antero-posterior section through humero-ulnar portion. Jessie R., age 70. Fibrous adhesions are present between the articular surfaces; also rarefaction of the cancellous tissue and sclerosis in the vicinity of the joint. Fibrous degeneration of bone is especially marked in the sclerotic parts. (*Specimen* S.C. 35—13.) (× 2·5.)

This process of resolution of bone and cartilage into fibrous tissue was present in most of the specimens from S.C. 35 microscopically examined; but in the left elbow a more advanced condition was found. The opposed surfaces of the humerus and radius were eburnated and sclerosed; small gaps in the surface bone were filled with granular-looking fibrous tissue (resolved portions of bone) which preserved the smooth unbroken level. The trabeculæ for some depth were composed of massed calcareous granules, stained purple by hæmatoxylin, and held together by a fine fibrous groundwork. Some of these degenerating trabeculæ still contained portions of bone having a blurred and granular appearance. Irregular lines and fissures were numerous in them, and evidently some of the trabeculæ were ready to break up into fragments and granular débris such as could be seen strewn in some places in the soft tissue. At one part on the surface of one of the bones was a long tract of crowded calcareous particles beneath a thin layer of degenerate cartilage, which was broken up into a fringe of short villi. There was no cohesion of the calcareous granules, and the tract had evidently resulted from disintegration of the calcified zone. In the sclerosed bone beneath it the trabeculæ presented the peculiarities just described. (*Fig.* 51.)

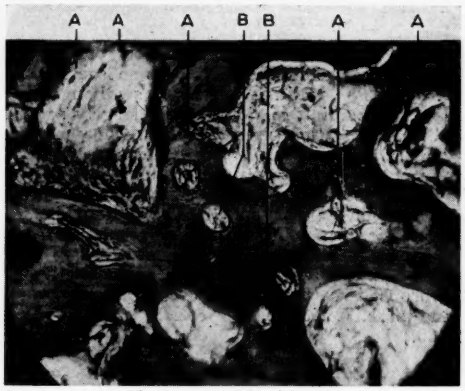

FIG. 50.—Fibrous degeneration of bone. Rheumatoid arthritis. A more highly magnified part of *Fig.* 49. The upper edge of the photograph adjoins the articular cartilage. The sclerosed subchondral bone undergoing fibrous degeneration. A, Points where fibrous resolution is well seen; B, Small spaces formed in the bone. Contents: fibrous tissue, connective-tissue cells, new vessels, a little fat, and some giant cells. The larger spaces are probably formed by the confluence of smaller ones. (*Specimen* S.C. 35—13.) (× 35.)

At another part of the section a striking appearance was produced by

several trabeculæ, parts of which were dead and parts alive. The dead parts were partially detached, and were composed of coarse fibrous tissue enclosing a number of swollen dead bone-cells. It was evident that in some parts of this joint, tissue death had occurred, and disintegration and not resolution into fibrous tissue was taking place. (*Fig.* 52.)

Different theories might be suggested to account for the peculiar changes met with in this case. The simplest would be that the vitality of the joint tissues was of so low a grade that reaction to toxic irritation was practically impossible, and slow degeneration, as evidenced by a reversion to a less highly organized form of connective tissue, resulted. In some places the tissue vitality was probably below the general low level, and at those points necrosis occurred. The histological evidence explains the absence of bony ankylosis.

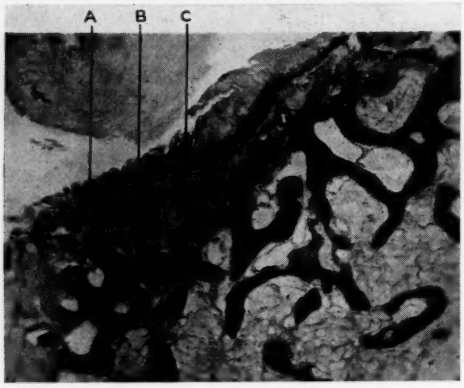

FIG. 51.—Rheumatoid arthritis. A degenerating articular surface from the same elbow as that shown in *Fig.* 49. The photograph was probably taken from the radius; the opposing bone shows very similar conditions. A, Necrotic cartilage; B, Layer of disintegrated lime granules, staining purple with hæmatoxylin—the calcified layer; C, Superficial trabeculæ undergoing granular disintegration: fibrous tissue in the medullary spaces, in which strands of calcareous granules, as well as débris, can be seen. The trabeculæ are crumbling at their edges and apparently resolving into delicate fibrous tissue, in which there are no cells. (*Specimen* S.C. 35—13.) (× 25.)

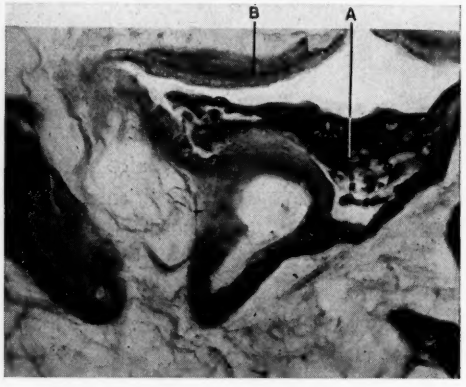

FIG. 52.—Rheumatoid arthritis ending in fibrous ankylosis. From the same section as *Fig.* 51. The radius. A, Dead swollen bone-cells in a portion of a trabecula largely separated into a space, possibly resulting around it within bone. The dead portion is largely degenerated so that only a thin infiltration of lime salts maintains its shape. B, Degenerated deep portion of the subchondroid bone layer, above which is the fibrous layer resulting from degeneration of its superficial portion, of the calcified layer, and of the articular cartilage. A fibrous ingrowth from the synovial membrane blends with it. (*Specimen* S.C. 35—13.) (× 50.)

That is obviously impossible between opposing bony surfaces undergoing necrotic disintegration or resolving into fibrous tissue, especially when, as in the latter case, the fibrous tissue between the bones is being augmented in the process.

The two cases represent two clearly defined forms of rheumatoid arthritis. Both are inflammatory, though only in the first case is the condition marked by migration of leucocytes to a notable degree; yet the absence of this unmistakable sign of inflammation in the second case is easily understood. Bony ankylosis was the usual termination in the first case, and failed in the second owing to the degenerative changes consequent on the low-grade vitality of the tissues.

A further insight into the nature of these conditions may be obtained by a study of the changes present in those cases in which the signs of osteo-arthritis and rheumatoid arthritis are combined.

Case 3.—A woman, age 76, is represented by nine specimens (Group S.C. 41) and in none of them is there bony ankylosis. The joint disease lasted twenty-five years, and its onset had been rapid. All the joints were involved in the first three weeks, and she was crippled in the course of a few months. After death a chronic empyema with a thick wall was found in the left pleura. It contained a pint of pus. Pure cultures of streptococci were obtained from the pus, and also from rabbits that died after inoculation with it. The hypertrophic manifestations were clearly of long standing. Marginal lipping was pronounced, and in the knees, where the different bones had been in contact, the articular surfaces showed flat plate-like elevations.

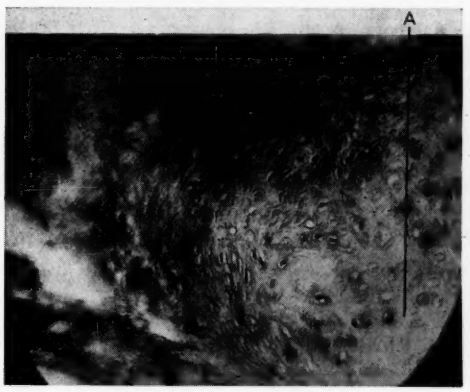

FIG. 53.—Rheumatoid arthritis. To show bone metaplasing into fibrous tissue. From the same section as *Fig.* 49. A, Bone. (*Specimen* S.C. 35—13.) (× 125.)

The microscope shows the principle seat of mischief to be where the cartilage joins the bone. The calcified zone and the subchondral layer of bone have been largely destroyed. A row of spaces is present at the line of junction, and they tend to spread along it and coalesce. The spaces have evidently resulted from the fibrous degeneration of the bone and calcified cartilage, and contain many stellate cells and vessels lying in a meshwork of fibrous tissue. By this process a considerable thickness of fibro-cellular tissue has come to lie between the cartilage and the cancellous bone-end, and the resolving edges of the adjacent trabeculæ and the deep fibrosing layers of the cartilage tend to blend with it. Patches of granulation tissue occur here and there within it, and many inflammatory cells are present in the medullary spaces below it. There is no sign of recent ossification, and as the articular surface is constituted by the superficial layers of the cartilage, and, where they have disappeared, by the fibrocellular layer, the fibrous form of ankylosis is in consequence assured.

The hypertrophic bone formations are the subject of degenerative changes equally with the rest of the articular surfaces—a circumstance that is strongly suggestive of their development before the ability to react had given place to degeneration.

A consideration of the foregoing facts points to the conclusion that it is the onset of degeneration that determines the fibrous form of ankylosis, and its essential feature is resolution of the articular bone and cartilage into fibrous tissue.

THREE CASES OF RHEUMATOID ARTHRITIS IN YOUNG PEOPLE.

The cases that furnished the material for the foregoing observations were either of late middle age or advanced in years. The cases that follow occurred in childhood or adolescence. Two, like *Case* 1, developed bony ankylosis; the other showed no such tendency and resembled *Cases* 2 and 3.

Case 4.—The disease began in Maud B. in a metacarpo-phalangeal joint when she was 18 years old. One hand and wrist and the feet and ankles next became involved. Both elbows and both knees were excised by Mr. Zachary Cope at the age of 30, but the left knee was not kept and there is some doubt as to the nature of the ankylosis present. Now at 37 years of age (1929) the left hip and shoulder, both wrists and ankles, various finger-joints, and the occipito-atloid and atlo-axoid joints are rigidly fixed, and bony ankylosis is probably present in some but not in all.

There was nothing definite in the history to account for the onset, but two years later some bad teeth were extracted, and an abscess of the breast was treated. There was a slightly rheumatic family history. The question of gonorrhœa was definitely eliminated in hospital. Amenorrhœa lasted from the twentieth to the twenty-seventh year, when a leucorrhœal discharge set in and has continued. Her periods are now regular.

The operations seem to have had a very beneficial effect on the course of the disease, which is apparently quiescent. The specimens (Group S.C. 28) include both elbows, in which all three bones are fused, and the right knee, in which the patella is ankylosed to the femur by bone.

Case 5.—Alice R, age 21, was 7 years old when her hands, wrists, and ankles, and then her knees, became swollen and painful. At the age of 9 they were 'set fast'. At 21 practically every joint in her body was involved, and wrists, elbows, knees, and hips were ankylosed. Movement in the small joints was limited. At 18 she had hæmoptysis. She never lost her cough. She had also a left purulent otorrhœa and offensive breath. She died at the age of 41 of tuberculous peritonitis and pneumonia, 'crippled, deformed, and phthisical'.

The specimens (S.C. 24) are two elbows showing bony ankylosis. They were excised when the patient was 21. Skiagrams show bony ankylosis of both knees, and of one superior tibiofibular joint.

No microscopical sections could be taken from these two cases.*

Case 6.—Elizabeth F. had her tonsils removed when she was 12 for frequent attacks of tonsillitis. Otherwise she was quite healthy till she was 13½ years old, when the joint affection began with pain in the right wrist and swelling of the knees. At the end of five weeks all her joints were affected, including the mandibular. She was an in-patient at several hospitals during some years, and at 17 was chair-ridden, with bent legs in which movement was very limited. At 18 she suffered from influenza and was again an in-patient at St. James's Hospital, Balham, where she remained six and a half years. When she was 21 both knees were excised by

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* The following case was under the care of my colleague, Mr. Edward Ward, of Leeds. It shows a certain similarity to *Cases* 4 and 5, but is a variation from the progressive and more usual type of rheumatoid arthritis :—

Percy B., age 13, a healthy-looking boy, suffered when 2 years old from some illness which was associated with pain in both legs. The legs gradually became flexed at a right angle, and when he was 5, were permanently stiff. Both knees and both ankles were ankylosed, but no other joints, and he had no pain. Mr. Ward removed wedges of bone from both knees, and sagittal sections disclosed intimate union between the femur and tibia in both. The patellæ were not ankylosed. (*Leeds Medical School Museum*, A 568.)

Mr. Zachary Cope, and in 1929 (age 27) her condition was much improved and the disease quiescent. She was chiefly inconvenienced then by the disabilities incidental to the much restricted movements of the different joints (shoulders, elbows, wrists, hands, hips, and ankles) which necessitated a reclining position.

The only specimen preserved is a sagittal section through the right knee in which all three bones are in close fibrous adaptation in a position of flexion (S.C. 51).

The microscopical examination shows that resolution into fibrous tissue is going on in the cartilage and bone forming the articular surfaces of all three bones. The degenerative changes are practically identical with those seen in *Case* 2, but inflammatory tissue is more in evidence.

No doubt in elderly people the tissues generally, including those of the joints, are much less robust than in youth, and ossification is naturally more active in the young. Consequently bony ankylosis may be expected to be more common in early life, and infrequent when the affection develops in middle life or old age. These juvenile cases show that the form of arthritis which a susceptible joint may develop in rheumatoid arthritis does not depend upon age alone, and the fact that in *Case* 6 the disease ran its course and reached its climax before the patient was 20 proves that fibrous degeneration of bone and cartilage is not a purely senile change.

(*To be continued.*)

IMPLANTATION OF THE URETERS FOR INOPERABLE VESICO-VAGINAL FISTULA AND ECTOPIA VESICÆ: A NEW TECHNIQUE.*

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The problem of devising a simple one-stage operation of implanting the ureters into the bowel has long been occupying my mind, for if the blood-urea estimation and general condition of the patient are fair, there would seem no adequate reason for failure, provided the technique is simple and within the means of the general surgeon or pure gynæcologist.

It is of course admitted that the results of a two-stage operation are excellent. The statistics of Fraser, the pioneer of this operation, Gow, and myself in India, and those of Coffey, Grey Turner, Jocelyn Swan, Nitch, and others in the West, all amply demonstrate that fact; but it seemed to me that one should be able to obtain Coffey's results, in the one-stage operation, if a simpler and more rapid technique could be discovered; for such an operation in the hands of a quick surgeon is called for in cases of ectopia vesicæ, inoperable and inaccessible vesico-vaginal fistulæ, carcinoma of the bladder, and certain gunshot injuries of the pelvis, and occasionally in post-radium bladder fistulæ.

HISTORICAL.

Between the years 1899 and 1903, Peters in Canada and Lendon in Australia were the first to publish successful cases of transplantation of the ureters. Since then, surgeons all over the world have endeavoured to improve the technique. In the early days of the operation, surgeons transplanted the ureter-bearing area of the bladder, with its valve mechanism, into the bowel, but their results were only successful in 50 per cent of the cases, owing to the fact that the physics of the living was not appreciated—that is, they failed to differentiate and separate the function of a sphincter from that of a valve, and failed to recognize that urine is delivered, after operation, from the duct of an organ which works under low pressure into a receptacle where the pressure is higher.

It is to Robert Coffey that surgeons owe the elucidation of previous failures, for he followed out Harvey's injunction "study and seek out the secrets of Nature by way of experiment". In 1908, when working with the Mayo brothers, investigating pancreatic surgery, he demonstrated that when the common bile-duct was transplanted by the direct method, it had become

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* Delivered at the All-India Science Congress, January, 1932.

enormously dilated when observed a few weeks later. This accidental discovery led him to the solution of the mechanism used by Nature when secreting fluid at low pressure into a cavity of higher pressure. Carrying his investigations further, he discovered the secret, so far hidden from surgeons and anatomists alike—namely, that in the case of such a tube as the ureter the duct first penetrates the muscle, then runs immediately beneath the loose mucous membrane for a distance before it emerges into the lumen of the bowel. This being so, in order to achieve success in such a case as implantation of the ureters, it was essential to devise an operation which copied the valve mechanism found in Nature. Experimenting on dogs, Coffey's logical deductions were completely justified, and within the next few years Dr. Mayo, giving all credit to the brilliant conception of Coffey, operated with success on a number of cases of ectopia vesicæ, dealing with one ureter at a time.

In 1925 Coffey, having done many of the two-stage operations of transplantation of the ureters which he had originally planned, became dissatisfied with the misery and delay entailed, and therefore commenced a series of experiments, using ureteric catheters as a means of conveying urine from the kidneys in order to obviate the risk that he had observed of an inflammatory exudate in the gut wall compressing the ureter and so predisposing to uræmia. After many trials and ups and downs of technique he evolved the admirable operation which was published in the *Annals of Surgery*, in June 1927, for bilateral transplantation of the ureters, and in November, 1929, he published an inspiring report of 20 cases operated upon by this method with only 1 immediate surgical death.

While all honour must be rendered unto Coffey for the original research work, it must not be forgotten that Sir Harold Stiles in Edinburgh, and later Grey Turner in Newcastle, from 1911 onwards, were performing a two-stage operation of implantation of the ureters, using a technique much after the fashion of Witzel's gastrostomy ; Grey Turner recording 17 cases with 4 deaths.

The first implantation operation in the Eden Hospital, Calcutta, was performed in 1912 on a girl of 14 suffering from exstrophy of the bladder. Briefly stated, Peters' technique was followed. After a stormy convalescence complete cure resulted. The patient was seen from time to time, and in 1926, when she was 28 years of age, she came to hospital nine months pregnant. Cæsarean section was performed with complete success.[1]

The admirable results reported by Fraser, in Madras, of cases of the two-stage operation for inoperable vesico-vaginal fistulæ inspired Gow and the writer to follow his technique, and our results of this two-stage operation are commendable. But for a long time it has seemed to me necessary to devise a simple operation with all the advantages of Coffey's method without its complicated technique—a method which would eliminate the dangers and disappointment of two anæsthetics on cachectic young or old patients.

The accompanying series of diagrams (*Figs.* 54–56) will not only explain the technique used in my first series of operations for bilateral ureteric implantation, but will make clear the two-stage operation to those surgeons who have as yet not attempted it, or have no desire to run the risk of transplanting more than one ureter at a time.

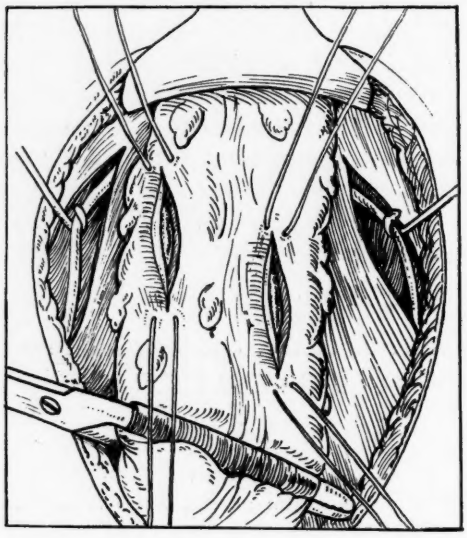

FIG. 54.—Before making the two gutters shown here on each side of the longitudinal band, the left gutter being at least 1 in. below the level of the right, long silk threads have been passed through the bowel muscle above and below the estimated incisions. Traction on these threads shown above permits the bowel wall to be held taut while the incision down to the mucous membrane through the muscle is made.

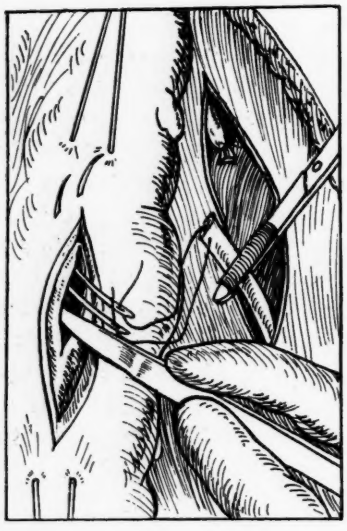

FIG. 55.—Catgut threaded at both ends with intestinal needles is passed through the lumen and wall of the ureter. The ureter is carefully laid in the gutter of the bowel and the two needles are passed through a stab wound made in the mucous membrane at the lowest portion of the gutter. The needles are made to emerge ¾ in. below the gutter and about ¼ in. apart. The two threads are then gently drawn upon until the cut end of the ureter is well through the stab incision. The catgut threads are then tied, so anchoring the ureter gently to the mucous membrane of the bowel.

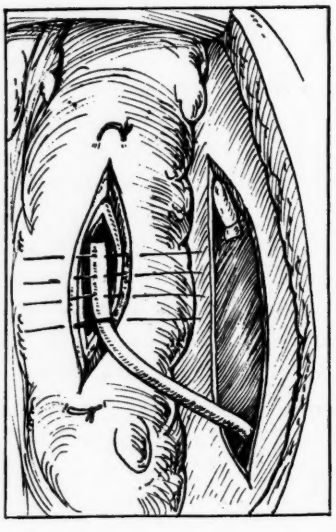

FIG. 56. — The ureter in position, four ligatures of catgut are passed from one side to the other through the peritoneum, muscle, and wall of ureter to the opposite side and then tied. The stab wound in the mucous membrane is separately sutured with three catgut sutures which pass through the ureter and the mucous membrane on each side, so preventing any back leakage from the bowel through the stab incision.

CASE REPORTS (*Group I*).

Case 1.—S., age 20, admitted for dribbling of the urine, subsequent to difficult childbirth one year ago. There is atresia of the vagina, which feels like a cartilaginous canal. The fistula is on the anterior wall, the size of one rupee. There is a history of one month's amenorrhœa. Blood-urea 0·015 per cent. General condition fair.

Both ureters were transplanted at one sitting, and except for twelve days' post-operative fever, recovery was uneventful.

At the time of operation she was seen to be pregnant. On April 3, 1931, she was re-admitted in labour and was delivered by Cæsarean section of a healthy child, leaving hospital fifteen days later.

Case 2.—H., age 18, history of still-birth and difficult labour eight months previously. The vagina admits one finger only, and in the vault of the vagina there is a fistula with a sharp fibrous margin through which the upper surface of the bladder projects. The pelvis is rachitic. General condition fair.

Both ureters were implanted on Sept. 1, 1930. The following day the general condition was good; temperature 100·5°; 23 oz. of urine had been collected from the rectum. The next day she passed 34 oz., on the third day 16 oz., and on the fourth day only a few ounces were passed. She rapidly sank into coma and died of uræmia that night. Post-mortem was refused, but there was no distension or peritonitis.

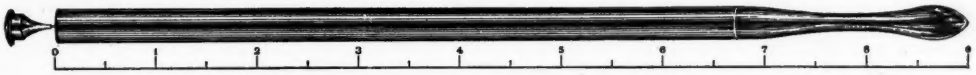

FIG. 57.—A full-size prostatic catheter from which the curved end has been removed and a screw bulb-end substituted. Total length 9 in.

I am convinced that the ureters in this case were compressed by an inflammatory exudate which dammed back the flow of urine, precipitating uræmia. For this reason I did not feel justified in further attempts to implant both ureters at one sitting until I could devise some method of getting over this difficulty of an inflammatory exudate compressing the soft ureter. During the interim therefore I went back to the two-stage operation and implanted many single ureters at a time without a death. But I was not satisfied, and determined to try again, using a means readily accessible to any surgeon—namely, the straight portion of a metal prostatic catheter, the curved part of which was cut off and a straight conical screw bulb substituted (*Fig.* 57). When all is ready the metal tube is passed through the stab incision down to the anus, and its canal is used as a tunnel to transmit the ureteric catheter to the rectum and anus without bother or kinking. (*Fig.* 58 will, I trust, make the following account intelligible.) But before proceeding I will describe the technique first used for one or both ureteric implantation operations.

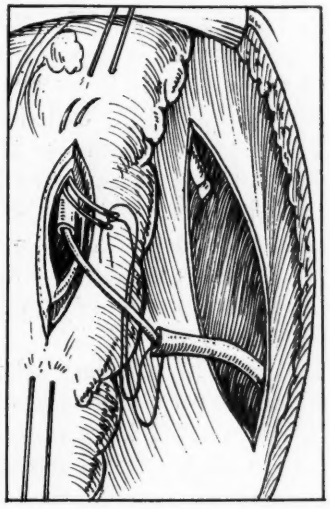

FIG. 58.—One ureter has been tied off close to the bladder and cut. As before, catgut threaded at both ends has been passed through the lumen and wall of the ureter. A ureteric catheter has been passed up the ureter for 5 or 6 in. The proximal end of the catheter is then passed down a straight silver tube, the proximal end of which will be seen emerging from the stab incision.

TECHNIQUE.

For nine days before operation the rectum is washed out daily with saline or weak permanganate solution. For the first three days the patient is given the following prescription four-hourly :—

Potas. Cit.		Glucose	℥iij
Mag. Carb. Pond. āā. gr. xxx		Aqua	℥j

The second three days 1 drachm of acid. sodii phosph. in water is given first thing in the morning, followed by 10 gr. of urotropine three times a day; the third three days the former alkaline mixture is taken.

Directly the patient is under the anæsthetic, an assistant very slowly perfuses 300 c.c. of a 20 per cent solution of glucose into the vein.

After opening the abdomen with the patient in the Trendelenburg position, the intestines are walled off in the usual way; the right ureter is quickly dissected out down to the bladder, leaving as much of its peritubular fascia around it as possible, and a piece of linen tape or a small blunt hook is passed under it. The ureter is not cut and made ready for implantation until the next stage of the operation is complete.

The sigmoid is held up by two silk threads which are inserted about 3 in. apart. A gutter is now made in the bowel wall 1½ in. long between the two silk threads, which are held taut. The handle of one's knife makes an excellent reflector of muscle and peritoneum. This stage of the operation takes only a few minutes.

The ureter is now caught up by a rubber-covered Kocher forceps, tied, and cut near the bladder, the distal end being touched with carbolic. The proximal end is brought up to the surface, and the pen-cut proximal lumen of the ureter is traversed with catgut, threaded with a needle at both ends, and a No. 12 flute-ended opaque catheter is passed for 5 or 6 in. up the ureter. The ureter is bound to the catheter by a single loop of 000 catgut.

A stab incision is made at the lower end of the gutter into the colon, and a straight short metal tube with a screw top is passed through it down to the anus. An assistant grasps it, unscrews the cap, and then the proximal end of the catheter is passed down the tube until the lie and line of the ureter are in the gutter previously made. *The metal tube is then pulled down and out of the rectum and reboiled ready for the other ureter procedure.*

In some of the early cases I had considerable difficulty in coaxing the metal tube down to the anus sufficiently far for my assistant to reach it with his fingers. I have now countered that trouble and source of delay by the simple procedure of passing into the rectum before the operation a medium-sized Kelly's cystoscope loaded with its blunt-nosed plunger, just as one would pass a proctoscope. When the stab incision referred to above is made in the bowel wall, my assistant directs the cystoscope towards the incision, the plunger is then removed and the metal tube containing the catheter is passed directly into the lumen of the cystoscope, and pushed through from above out of the proximal handled end of the cystoscope. This manœuvre is simple and quick and has given great joy since its discovery.

The double threaded catgut needles are then passed through the stab incision to emerge ¾ in. below the gutter and about ¼ in. apart. They are

drawn upon, thus bringing the ureter down through the stab wound into the bowel; the catgut threads are then tied, thus loosely anchoring the ureter to a point at least 1 in. below the stab incision. Three or four interrupted fine catgut sutures are then passed from one edge to another, picking up the peritoneum, muscle, and ureter in order to close the incision and secure the position of the ureter. The edges of the stab incision are brought in close contact by fine catgut which traverses the wall of the ureter, thereby further anchoring the ureter and preventing back leakage from the rectum. The remaining portion of the catheter-containing ureter in the gutter is then covered by a continuous Lembert suture up to the site where the ureter emerges from its bed under the initial incision in the peritoneum as in Coffey's illustrated article.

Exactly the same technique is adopted on the left side with the left ureter except that the incision in the bowel wall is on the left of the longitudinal band, and about 1 in. below the level of the right gutter.

The two subperitoneal tunnels from which the ureters were brought up are now closed with catgut, and after a look round to see there is no oozing or any retroperitoneal hæmatoma forming—all towels are removed—the omentum is brought down and the abdominal wall is sutured in the usual way. No drainage is used. In all eight cases now to be recorded the urine began to flow at once from each catheter. In order to prevent kinking or pressure on the catheters they are threaded through a short flatus tube which is passed through the anus. The ends of the catheters are inserted into a measured quantity of carbolic solution.

CASE REPORTS (*Group II*).

Case 3.—I., age 22, history of one difficult labour. Now almost complete atresia of the vagina, the fistula being visible at the top of the canal, which admitted the little finger only. There was in addition a stone in the bladder the size of a hen's egg and a recto-vaginal fistula high up. Blood-urea 0·022 per cent, Hb. 55 per cent, R.B.C. 2½ million. On Aug. 26, 1931, the stone was removed after incising the vagina.

A fortnight later, under pernocton and ether anæsthesia, both ureters were transplanted into the rectum by the method described. The patient ran a temperature for twenty days but made an uninterrupted recovery, the recto-vaginal fistula being successfully operated upon on Nov. 13, 1931.

Case 4.—B., age 20, admitted on Sept. 4, 1931. History of one difficult labour. The whole of the base of the bladder and urethra have sloughed away, leaving a large gap surrounded by dense fibrous tissue. The patient was pregnant two months. Blood-urea 0·02 per cent, Hb. 50 per cent, R.B.C. 3 million.

Transplantation of both ureters, as above, was performed on Sept. 14. Except for slight fever for the first ten days and the fact that the patient normally aborted on the eighth day after operation, recovery was uninterrupted. She left hospital cured on Oct. 15.

Case 5.—B., age 40, admitted on Oct. 7, 1931. History of three difficult labours —the last resulting in the formation of a fistula, the size of a rupee, at the junction of the cervix and bladder, surrounded by dense scar tissue. Blood-urea 0·04 per cent, Hb. 70 per cent.

On opening the abdomen there was a large right tubo-ovarian hydrops. This was first resected. Both ureters were then transplanted as above. The patient was in excellent condition, passing urine plentifully for the first three days, but on

the fourth day she suddenly became cyanosed, coughed up blood, and died of pulmonary embolism.

Case 6.—R., age 18, admitted on Oct. 8, 1931, with a large fistula involving the urethra and bladder the result of difficult labour with dead child eight months previously. Blood-urea 0·036 per cent, Hb. 70 per cent.

Careful vaginal dissection failed to approximate the edges. Laparotomy was done on Dec. 7. Both ureters were implanted, using opaque catheters. An interesting feature of this case was the presence of two ureters on the left side lying collaterally. The upper ureter, being twice the calibre of the lower one, was the one implanted. This condition of double ureters is recorded by both Grey Turner and Thomson-Walker. Recovery after ten days' slight pyrexia was uneventful, both implanted ureters draining perfectly. There was just the scantiest wetting of the bedding from the vagina, owing to the lower ureter slightly functioning. It is probable that this extra ureter will cease to function; if not, it will be implanted separately later. The patient is walking about and quite happy on the fifteenth day after operation. On Feb. 17, 1932, the extra ureter was dealt with. She left hospital cured on March 1.

Case 7.—F., age 18, admitted on Jan. 11, 1932, with an enormous rigid hole at the base of the bladder due to difficult labour one year ago; the baby was born dead. Hb. 45 per cent, blood-urea 0·025 per cent. Transplantation of ureters on Jan. 25. Urine drained well from the start. Had double rise type of fever for three weeks which reacted to urea-stibamine and was shown to be due to kala-azar. She left hospital happy on March 5.

Case 8.—A., age 19, admitted on Feb. 2, 1932, with an atresic vagina and unapproachable fistula due to labour eight months previously. Hb. 50 per cent, blood-urea 0·63 per cent, R.B.C. 2½ million. Under pernocton and ether both ureters were transplanted on Feb. 15. Recovery uneventful. Discharged cured on March 11.

Case 9.—S., age 30, admitted on Feb. 19, 1932. Her condition was the same as the previous case, and has existed since her last child was born dead twelve years ago. Hb. 46 per cent, blood-urea 0·03 per cent. Operation as before, but on the third day the temperature went up to 105° and remained high despite all treatment. She died of uræmia on March 19. Post-mortem refused.

Case 10.—A., age 28, admitted on March 12, 1932, for a fistula admitting two fingers at the top of a lacerated vagina. She had had twelve labours, all at full term, and all the babies had been born dead. The fistula had only occurred after the last childbirth. Both ureters were implanted on March 21. For the first eight days all went well, but the patient then grew very boisterous. The temperature became subnormal. She passed only 12 oz. of urine for the next two days, and then sank into coma, dying on March 31. At autopsy, both kidneys were large, sacculated, and full of pus, from which *B. coli* and staphylococci were cultured.

OPERATION COMMENTS.

Some surgeons insert a suprapubic drain for twenty-four hours. The experience gained from a large number of both classes of operations convinces me that this is not necessary, all the patients having healed by first intention. Grey Turner[2] implants both ureters on the right of the colon and into the longitudinal muscular band, one 2 in. above the other, the left ureter being passed under the colon. I think this must be a more difficult procedure and conducive to oozing.

In some cases the ureter is greatly dilated owing to obstruction by old inflammatory tissue in the parametrium. When inserting the catheter in these cases it is wise to run a purse string round the end of the ureter so as to

grasp the catheter lightly. In order to secure good vision in the field of operation a large Doyen retractor is better than anything else. Before closing the abdomen the uterus should be ventro-suspended to prevent adhesion of its posterior surface to the bowel and subsequent pelvic disorders. All patients should be warned in the event of pregnancy to attend an antenatal clinic and come to hospital in their eighth month for Cæsarean section at term (*see Case* 1).

All patients, whether the technique be that of a one- or two-stage operation, run a temperature for ten or twelve days afterwards, presumably owing to mild pyelitis. The first case in 1912 of ectopia vesicæ, with Cæsarean section in 1926, illustrates the small chance of permanent injury to the kidney, if that organ is healthy at the time of operation. All patients are given copious alkaline drinks and fruit juice after operation. In both operations castor oil is given on the fourth morning.

In the two-stage operation, where no catheter is used, the rectal tube is removed about the fifth day. In the one-stage operation, using ureteric catheters, they are drawn out on the fifteenth day if they have not come out before. Coffey tightly binds the distal half-inch of his ureters to the catheters in order to prevent intestinal infection reaching the ureter, but such a procedure, I think, must make difficult the removal of the catheters. Moreover, by causing sloughing of the ligated portion of the ureter it would seem to me to risk defeat of the very object for which the catheters are tied in.

In one of my two-stage operations there was quite severe arterial bleeding, a few hours later, from the rectum, no doubt owing to my having cut a branch of the hæmorrhoidal artery. The hæmorrhage was checked by iced water enemata containing adrenalin.

In future cases I intend to follow Wilkie of Edinburgh's custom, and give nucleic acid and *B. coli* vaccine to all patients prior to operation. Moreover, I am of the opinion that patients in the Tropics, anæmic and debilitated, over the age of 25, will not stand the strain on both kidneys that double implantation demands, and therefore I shall in such persons use the same rapid easy technique on two separate occasions.

Ureteric catheters are not expensive, but it is important that they should be new, and at least No. 12 to No. 18 in size, with flute ends. The use of catheters eliminates the dangers of inflammatory exudate or peristalsis compressing the soft non-tensile ureter during its course through the gutter valve in the bowel wall.

A great many of our fistulæ patients are cases with small round pelvis or osteomalacia, in whom the great damage to the cervix, bladder, and vagina in the course of long unaided labours in distant villages is easily explained. But despite the fever, sloughing, and atresia that occur it is remarkable to observe in practically all, that on opening the abdomen, the uterus is mobile and the Fallopian tubes are normal and patent. Again, it is interesting to note that in two cases (*Cases* 1 and 4) both patients had become pregnant just before operation despite large high vesico-vaginal fistulæ. In this connection it may be of interest to note that on April 16, 1932, a Mrs. C. was admitted for confinement who had been born in this hospital eighteen years previously with an imperforate anus and congenital high rectovaginal fistula. Cæsarean section was performed, and she left the hospital with her infant on May 5.

Clinically it has been a pleasure to see the joy and growth in weight of these patients as they come to realize that the misery of their previous condition has been or will be alleviated. I have found that after two or three weeks the anal sphincter becomes so alert that they can retain quite a surprising quantity of urine in the descending colon for three to four and a half hours by day and at least six hours at night.

It is admitted that these eight case reports of successful implantation of both ureters, using opaque catheters and a metal tube to transmit the catheters to the anus, is but a small number from which to draw conclusions. But it is hoped that surgeons who have been dismayed by the elaborate technique of Coffey will give the method a trial, commencing, if need be, with one ureter and two separate operations; later, when they have learnt the technique, perhaps they will be persuaded to implant both ureters at one sitting; for cases of carcinoma of the bladder and cervix or vesico-vaginal fistula are often in a pitiable condition of pain and suffering.

I feel that any technique which is simple and within the means of all surgeons, and especially those attached to a woman's hospital in the Tropics, will perhaps commend itself. The average time taken for double implantation has been one hour and a half.

It will give readers some idea of the frequency and, I might almost say, the malignancy of some of these fistulæ seen in the East when one relates that during the last ten years—that is, before we adopted transplantation of the ureters in desperate cases—my Registrar, Dr. R. R. Roy, has collected the case sheets of 104 consecutive patients admitted into this hospital with vesico-vaginal, uretero-vaginal, cervico-vesical, or urethro-vaginal fistulæ. He finds that out of these 104 operated upon, some of them multiple times, 55·8 per cent only were cured, 40·4 per cent failed, and 3·8 per cent died.

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RESECTION OF THE PRESACRAL NERVE IN UROLOGICAL CASES.*

By GORDON S. FOULDS, Toronto.

During recent years neurosurgery has been making rapid progress, and this is particularly true of surgery of the sympathetic nervous system. Little attention, however, has been paid to the application of this branch of surgery to the urinary bladder. Until quite recently our knowledge of the more exact anatomy of the nerve-supply of the bladder was sadly deficient. While most standard text-books of anatomy deal very inadequately with the subject, the French surgeons and anatomists have led the way, both in anatomical investigations and the application of anatomical knowledge to surgical intervention. The studies of Latarjet and Bonnet in 1913, and subsequent extensive work by Laux, together with clinical reports by Pieri and others, were largely disregarded until the work of Learmonth, who has done much to further clinical application of the anatomical data provided by the French investigators. In this more recent work many clinical observations have been possible, which lead us to a better understanding of the neurophysiology of the bladder.

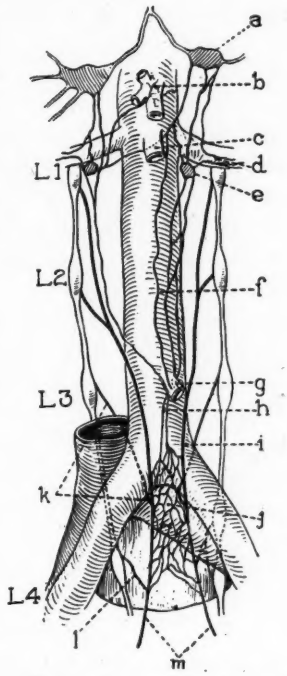

Fig. 59. — Connections of the presacral nerve. *a*, Semilunar ganglion; *b*, Cœliac plexus; *c*, Superior mesenteric artery; *d*, Renal plexus; *e*, Renal ganglion; *f*, Intermesenteric plexus; *g*, Inferior mesenteric artery; *h*, Middle root of presacral nerve; *i*, Left lateral root of presacral nerve; *j*, Presacral nerve; *k*, Branch from third lumbar ganglion; *l*, Branch from fourth lumbar ganglion; *m*, Hypogastric nerves. (*After Laux.*)

Micturition depends upon a very complex reflex, involving both the bladder itself and the internal and external sphincters. There is a diphasic neuromuscular mechanism; the filling phase, which is involuntary, requires the relaxation of the bladder with contraction of the internal sphincter, while the emptying or detrusor phase is under voluntary control and involves relaxation of the sphincter and contraction of the bladder muscle. The bladder and its sphincters receive their nerve-supply from three sources—the thoracolumbar outflow of the sympathetic system, through the hypogastric nerves; the sacral autonomic system by the pelvic nerves or nervi erigentes; and from the somatic centres in the sacral part of the cord through the pudic nerves.

* Read before the Surgical Section of the Academy of Medicine, Toronto, on Oct. 20, 1931.

The hypogastric nerves have their origin in the presacral nerve described by Latarjet. This nerve is composed of three roots. Fibres from the first and second lumbar ganglia of the sympathetic chain on either side form the two lateral roots, which converge to unite in front of the body of the 5th lumbar vertebra; they are pre-ganglionic and medullated. As these lateral roots converge they are joined by a third or middle root, which is made up of post-ganglionic, non-medullated fibres from the network of sympathetic nerves lying in close relation to the front and sides of the aorta known as the intermesenteric plexus (*Fig.* 59). These three roots unite in a varying manner to form the nerve bundle which descends parallel to the median line and crosses on the left common iliac vein, after which it follows the anterior surface of the sacrum. This bundle may be compact or separated, giving a fenestrated appearance. At a distance of 4 to 6 cm. the presacral nerve divides into the two hypogastrics, which pass into the pelvis to join the hypogastric ganglia. The inferior mesenteric ganglion, so constant in lower animals and frequently referred to in writings on bladder physiology, is usually absent in man, and when present is represented by a thickening in the intermesenteric plexus about the root of the inferior mesenteric artery. The fibres from the lumbar ganglia descend, as previously described in the lateral roots of the presacral nerve.

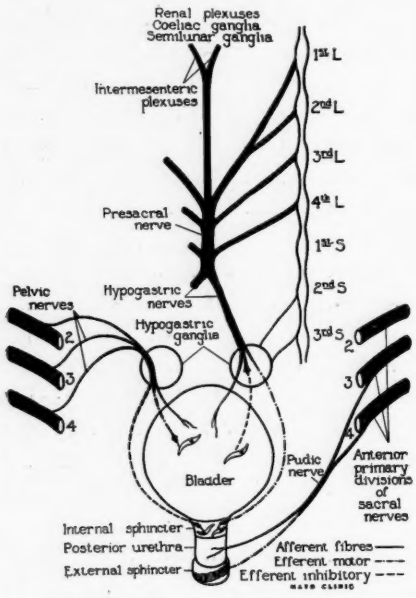

FIG. 60.—Anatomical diagram of nerves to the bladder. (*After Learmonth.*)

The pelvic nerves are composed of fibres from the anterior primary divisions of the 2nd and 3rd or 3rd and 4th sacral nerves. These are pre-ganglionic medullated fibres and pass through the hypogastric ganglions to synapse with the nerve-cells of the ganglionated network of nerves in the bladder wall.

The third pathway is through the deep perineal branch of the pudic nerve which supplies motor fibres to the compressor urethræ muscle and sensory fibres to the deep urethra. This nerve has its origin in the 3rd and 4th sacral segments of the cord.

The filling phase of the bladder function is dependent on the motor impulses transmitted from sympathetic outflow by way of the presacral and hypogastric nerves. This nerve pathway transmits efferent motor stimuli to the internal sphincter and motor inhibitory impulses to the bladder muscle. It also carries afferent fibres to the bladder which transmit sensory impulses, notably those of overdistension of the organ with suprapubic discomfort. This was noted by Head and Riddoch, who demonstrated that pain set up by the ineffectual contraction of an .overdistended bladder in endeavouring to overcome an obstruction at its outlet is referred to the 11th and 12th thoracic and 1st lumbar dermatomes, the somatic nerves of which arise in

the same spinal segments as some of the pre-ganglionic connections of the hypogastric nerves. This nerve contains in addition vasoconstrictor fibres to the bladder. (*Fig.* 60.)

The emptying or detrusor phase of bladder action is under control of the pelvic nerves, which carry efferent motor stimuli to the detrusor muscles and motor inhibitory impulses to the internal sphincter. Afferent fibres from the bladder through these nerves represent a large proportion of the sensory fibres from the bladder.

Having reviewed briefly the nerve-supply to the bladder with reference to its function, I should like to report a case illustrating the application of some of these data.

History.—Mr. I. M., age 22, was admitted to St. Michael's Hospital on Dec. 2, 1930, complaining of loss of bladder control of fifteen months' duration. On Sept. 24, 1929, while intoxicated, he had fallen from a third floor window and sustained a compression fracture of the 2nd lumbar vertebra. Immediately following the accident he received hospital treatment and was placed in a plaster cast for three months. Neurologic examination at this time is said to have shown some paresis of the legs, but no loss of any localized movements, and sensory changes in a saddle-shaped area about the anus and scrotum in the area of the 3rd to 5th sacral segments, where there was an absence of sensation to pain and touch. There were no sensory changes in the legs or back.

He was unable to pass any urine and required to be catheterized regularly during the period in which he remained in the cast. Shortly after being allowed up he found that he was able to void by sitting down and pressing with the hands on the lower abdomen. He had no desire to urinate at any time, but felt pain suprapubically when the bladder became overfull. On such occasions the bladder could be felt as a pyriform mass in the lower abdomen, reaching nearly to the umbilicus. At first there was little or no difficulty in retaining urine, but later there was definite overflow incontinence at night or when careful attempts to empty the bladder partially during the day were neglected. These conditions continued up to the time of admission. Immediately after his accident there were no erections, but after several months the power of erection gradually increased until it had been fairly normal for over three months.

On Examination.—The patient was a well-nourished white male. General examination was normal except for the presence of a mass in the lower abdomen extending to within an inch of the umbilicus and for a good deal of excoriation about the external genitalia due to dribbling of urine. After voiding as described above it was found that there was a residual urine of 28 oz. Catheterization was painful. The deep urethra was very sensitive.

Rectal examination showed poor tone of the rectal sphincters. The prostate was normal. Sensory changes were as previously noted. The bulbocavernosus reflex was present but not very active. Examination of the urine showed it to be acid; specific gravity, 1022; albumin, trace; sugar, negative; microscopic examination showed the presence of a moderate number of pus cells. The white cell-count was 9200; blood-urea nitrogen was estimated as 12·2 mgrm. per 100 c.c. of blood. X-ray examination of the lumbar spine

showed a fracture-dislocation of the 2nd lumbar vertebra, while films of the urinary tract showed no evidence of stone or other abnormality. Cystoscopic examination was carried out under general anæsthesia. The internal sphincter was in spasm. The bladder contained 18 oz. of residual urine. Inspection of the bladder showed a general diffuse chronic cystitis. There was a moderate degree of trabeculation, seen particularly in the dome. Both ureteral orifices were normal in appearance and spurted clear urine. Ureteral catheterization with divided functional test and pyelograms showed no abnormality in the upper urinary tract.

Diagnosis.—A diagnosis of 'cord bladder' due to injury to the lower segments of the cord (3–5S.) was made.

Discussion of Case.—Taking into consideration the site of the injury, it is obvious that the sympathetic innervation of the bladder was not damaged. The hypersensitive posterior urethra, together with the weak but nevertheless present bulbocavernosus reflex, was taken to signify an intact pathway by the pudic nerves. The absence of any desire to urinate, and an inability to urinate satisfactorily, in view of the foregoing observations, corresponded with the localization of the injury and indicated serious interference with the sacral autonomic supply to the bladder by way of the pelvic nerves. However, in view of the observation that by taking time for relaxation of the sphincters and by the aid of the accessory muscles, partial emptying of the bladder was possible, and giving consideration to the fact that the power of erection had partially returned, it seemed reasonable to assume that there was not a total absence of impulses transmitted by the pelvic nerves, and that what really mattered was a lack of balance between the sympathetic and sacral autonomic innervation, the sacral autonomic normally being the positive or active force, while the sympathetic acts as a 'brake'. Now the 'brake' seemed to be too strong for the continued action of detrusor forces. Accordingly it was decided to interrupt the sympathetic innervation by resecting the presacral nerve and so remove its 'brake' action.

Operation (Jan. 5, 1931).—Under general anæsthesia a mid-line incision was made, extending from the umbilicus to the symphysis pubis. With the patient in deep Trendelenburg position the small intestines were packed up and the sigmoid to the left, thus bringing to view the bifurcation of the great vessels and the promontory of the sacrum. The posterior peritoneum was incised for about $1\frac{1}{2}$ to 2 in. over this area and retracted gently. The two hypogastric nerves were isolated with little or no difficulty and severed just below the level of the promontory of the sacrum, and then followed up to their origin in the presacral nerve. This nerve was sectioned about $\frac{3}{4}$ in. above this junction, at a point where it crosses the left iliac vein. The posterior peritoneum was now closed, the intestines allowed to resume their former position, and the abdomen closed. An indwelling catheter was fastened in position and the patient returned to bed.

Post-operative Course.—Immediately after operation the patient was given 0·12 grm. of acetylcholine twice daily intramuscularly. This drug is a specific stimulant of those structures innervated by the sacral autonomic system. In addition to this the bladder was washed regularly with 1–8000 potassium permanganate solution.

The indwelling catheter was removed on the tenth day. On the subsequent two days residual urine was found to be 15 and 12 oz. respectively. On the twelfth day a right-side acute epididymitis developed and no further estimation of residual urine was undertaken for ten days. At the end of this period residual urine was still 10 to 12 oz., so on Jan. 27 the indwelling catheter was again replaced for two weeks. After this period the residue was about 8 oz. Catheterization was done twice a day with gradually decreasing residue, until during March this was reduced to 1 or 2 oz. The man remained under observation until April 16, when he was dismissed from hospital with a residual urine of 2 oz.

When last examined, nine months after the operation, his chief difficulty was extreme constipation. When the constipation was very bad there was still difficulty with the bladder, at which time there was from 4 to 8 oz. of residual urine. Now, even if the bladder is allowed to become distended there is no suprapubic pain, proving the contention of Head and Riddoch that the sensation of overdistension is transmitted by the presacral nerve. It was also found that catheterization was not particularly painful, confirming the observation of Learmonth that the presacral nerve supplies some sensory fibres to the posterior urethra.

At the present time, under favourable conditions the patient can void fairly satisfactorily while standing, and on most occasions while sitting can empty the bladder to within 2 to 4 oz. He now has to depend entirely on the external sphincters for control. This is not always satisfactory at night, as urine frequently leaks during sleep. During the day control is generally satisfactory, but with over-filling of the bladder or fatigue, dribbling takes place. There is no question now of retention with overflow.

I think I may safely say, first, that this man is more comfortable; secondly, that residual urine has been reduced from 20 oz. or more, with distension and overflow—the usual condition—to 2 to perhaps 6 oz. Sphincter control is not well exercised, but I believe might be better with greater co-operation.

Pieri was first to point out the possibility of relieving the pain in some bladder lesions by section of the presacral nerve. In 1926 he performed the operation to relieve the distress from a painful cystitis. Viannay reported a further case the following year. Learmonth has made a preliminary report on four cases of this class. Three cases were suffering from a panmural cystitis, commonly spoken of as 'Hunner's ulcer', and the fourth was one of tuberculous cystitis, persisting nine years after a nephrectomy for tuberculosis, no tubercle bacilli being demonstrable in the urine at the time of the sympathectomy. Learmonth gives two reasons for attempting these operations: First, to relieve pain. This object was accompanied with a fair degree of success in three cases. Secondly, he is of the opinion that a more curative effect may be obtained by the vasodilatation brought about by the sympathectomy. The cases reported were too recent to make a definite statement as to what extent this hope was realized. Further, it cannot be claimed that all vesical pain can be controlled by presacral neurectomy.

Learmonth also reports two cases of sympathetic-parasympathetic

unbalance, in principle similar to my case. One was due to a residual myelitis of the conus. In both he obtained good results.

The number of cases in which this operation is applicable will naturally be small, but I believe it to be indicated in certain selected cases of unbalance between the sacral autonomic and sympathetic outflow, and in certain painful conditions of the bladder, particularly those associated with hypogastric pain with spasm of the internal sphincter.

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EIGHT LETTERS OF JOSEPH (LORD) LISTER TO WILLIAM SHARPEY.

By C. R. RUDOLF.

These eight letters written by Joseph Lister to William Sharpey were recently found by me in a second-hand bookshop in London. The bookseller was unable to say where he had obtained them. The first six letters submit Lister's ideas for Sharpey's criticism before offering his views to the Royal Society. These views, being approved by Sharpey, were embodied in various physiological papers which became well known to the scientific world. They led to Lister's recognition as an accurate and careful exponent of the processes of inflammation which had long exercised the minds of such thoughtful surgeons as Travers and Paget.

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11 Rutland Gate,
Edinburgh,
16*th November* /57.

My dear Dr. Sharpey,

I have received my paper for revision prior to printing in the transactions, and along with Mr. Stokes'[1] note was enclosed a report by Mr. Paget[2] on the paper. Of this I enclose a copy in case you may not have seen it; and now venture to trouble you for your advice on some points in Mr. Paget's suggestions, seeing that you are the "communicator" to whom he refers as the person through whom his suggestions should be conveyed to me: not to mention that your past kindness throughout this business, would have emboldened me to inflict myself upon you once more, even had you not occupied the position referred to.

First I must say that I am much impressed with Mr. Paget's care and candour in the matter, and, considering the position he occupies with regard to the subject, I think his remarks exceedingly handsome, and am much gratified by them. Mr. Goodsir[3] also mentioned to me his having read the paper and endorsed it with his recommendation for publication, and he too spoke in terms, I fear, considerably flattering regarding the production. I will now speak of each of Mr. Paget's suggestions and allude at the same time to Mr. Goodsir's remarks on the same points.

First as to omitting the account of the theories of previous writers. Mr. Goodsir also said, before he knew of Mr. Paget's report, that he thought the paper would be more classical without the historical sketch; although at the same time he said he thought it "excellent", supposing it necessary. But he did not think any such thing was required for a paper for the Royal Society. I am quite willing on these grounds that this part should be

omitted, although, for the sake of the *general* reader, I should have preferred its being retained. I shall be guided entirely by you on the point. When writing this I do not know how much discretion is left to me. Mr. Stokes says " you will probably see reason to adopt " his (Mr. Paget's) " suggestions ".

As to the second part which Mr. Paget recommends me to blot out, viz., the discussion of vital affinities, etc., in the second section, Mr. Goodsir does not appear quite to agree with Mr. Paget. The former observed, " that is the opinion of a London man ". Now, unless you have seen the paper you could hardly form a judgment without my writing at greater length than would be expedient; but I would ask you if there would be any objection to my retaining the mention of the observation of the absolute stillness of the blood in the capillaries and veins between the pulses in small frogs under the influence of chloroform. The fact that this state of things may continue for an unlimited time without any accumulation of corpuscles occurring in the vessels appears to me important, as the heart, though exceedingly weak, is then obviously the only cause of the movement of the blood. I would very much curtail my remarks on the bearings of the observation. It is not only in Edinburgh that persons think the vital affinities a cause of the blood's movement: witness Dr. Carpenter:[4] and here the opinion is rampant that [the] said affinities are the chief cause? As to the discussion on the mode in which the constituents of the liquor sanguinis required by the various tissues for the purpose of nutrition find their way out of the capillaries, this I confess is very hypothetical and I am quite willing to expunge it. But it is not long, and, if there be no objection, it might be printed, and then if you thought well I would strike it out of the proof.

With regard to the expression about " paralysing " the concentrating and diffusing forces of the pigment cells, I feel seriously at a loss to know how to find a different mode of conveying my meaning. The facts are simply these. In the state of health the pigment cells exhibit the functions of concentration and diffusion of the granular portion of their contents under obedience to nervous influence. Also when a healthy limb is amputated concentration of the pigment (if previously diffused) takes place to the full degree and the pigment remains concentrated for some hours, after which a certain amount of diffusion again occurs, but if certain agents be applied to a portion of the web the pigment in that part ceases to manifest these functions. Not only is this the case when the limb is in connection with the body, but also when severed from vascular and nervous connection with the trunk. If the agent be applied to part of a dark limb just amputated the post mortem concentration does not occur in that part and again if the agent be applied when post mortem concentration is at the full, the subsequent diffusion does not take place in the spot operated on. In short, these functions of the pigment cells not only cease to vary in obedience to nervous influence, but also fail to exhibit the changes that ensue sooner or later after blood has ceased to circulate through the part: of which the post mortem concentration seems comparable to the post mortem rigidity of muscles. Now since I began to write this sentence I have been troubled with a difficulty that never occurred to me before, and which, I dare say, may be what Mr. Paget feels: and reflection upon this has detained this epistle two days. When speaking of

" paralysis of the concentrating and diffusing powers ", I convey the meaning that these powers not only cease to *vary* but cease to operate at all. Now I admit that this is by no means " proved " in my paper as it stands. There is good reason, I there state, to believe that the concentrating and diffusing forces are both in constant antagonistic operation, both being equally balanced except when either diffusion or concentration is occurring. Now all that I have shewn is that in the irritated part neither diffusion nor concentration occurs, and it may therefore be fairly argued that this does not prove more than that those forces cease to *vary* in *relative* amount under nervous influence, an effect which might result from paralysis of the extremities of the nerves, while the forces in the cells remained unaffected. But the consideration of all the facts with which we are acquainted regarding these pigment cells, including some which I have observed since the paper was written, seem to me to *prove* that the *cells* are affected independently of the nerves, and that this affection is one of paralysis. Now I am truly sorry to inflict this rigmarole upon you, but do not see how I can help it; as I do not wish to run counter to Mr. Paget's opinion, and yet should be very sorry to give up the use of the term " paralysis " unless obliged to do so. You will remember that the German observers Brücke,[5] Wittich,[6] and Harless,[7] found that in the tree frog and the esculent frog concentration of the pigment resulted from the stimulus of light, from psychical excitement, from stimulation of the spinal cord, from stimulation of a branch of nerves leading to a particular portion of skin and from other stimuli. These observations tended to shew that concentration was the condition induced under the influence of stimuli applied through the nervous system, and that diffusion was the state with which the cells passed when left undisturbed by such stimuli. I have made similar observations on the frog of this country, which, however, is not nearly so sensitive to local stimulation, and I have also added the following fact lately. If I cut through all soft parts of the middle of the thigh except the artery, vein and nerve, the circulation goes on as usual and the leg retains the same colour as the rest of the body. But if I now cut through the nerve, the legs immediately become darker (if previously pale) and in a few hours the legs become black from full diffusion of the pigment, while the rest of the body may remain pale yellow. This very curious result is not *constant*, apparently in consequence of its being impossible to divide all the nervous filaments leading from the trunk to the limb, as proved by the fact that the arteries after a longer or shorter time regain their power of contraction, which is lost permanently if the brain and cord is removed. I have however induced this strange contrast of colour in three cases in which I performed the above operation. This experiment confirms the view that diffusion is the condition which occurs when nervous influence is removed.

An experiment by Wittich on the tree frog must be mentioned as illustrative of the same principle. He found that oil of turpentine when applied to a *moderately* dark tree frog caused the spot to which it was applied to grow pale, but after the oil had been removed the part not only returned to its former colour but became darker than other parts: the secondary diffusion of the pigment appearing comparable to the secondary dilatation of arteries after contraction from stimulation. So strongly did these facts impress the

German authorities that they considered them proof positive that concentration was the result of *contraction* of a cell and diffusion a relaxation of such contractile cell. Now I have shewn as you know, that this view is incorrect, as the cells do not change in form or size, but the pigment granules undergo changes of place in the fluid contents of the cells. And I have in the last few weeks made further observations on these curious processes, which shew that they are more complex than I had supposed. I have observed a cell in which slight diffusion was going on, and saw that the granules were moving in a very complex manner: one granule would be moving from the central dark mass of pigment while another was passing towards it. Sometimes a number of granules would start off together; then stop and some of them return, etc., etc. Shewing that diffusion is the aggregate result of a complicated set of movements of granules some towards, others from the centre. Sometimes one saw a single granule start off from the central mass and return to it after a circuitous course, as shewn in the diagram [omitted], where (a) is the central black mass of concentrated pigment, and the dotted line represents the course of a granule. Such movements reminded me of what one sees sometimes within the cells of vegetables.

This description of the movements of the granules you will say is a digression from the argument, and so it is, but I wish to mention it to you, in order to let you know that I have made some further progress. But, however complex the action of concentration and diffusion be, there nevertheless remain the facts shewing that concentration is what occurs in the cells when stimulated through the medium of the nerves, and diffusion is that which takes place when the influence of the nerves is withdrawn. Hence it appears to me clear that, as an agent which induces inflammation causes the pigment cells to remain in the state in which it found them (say that of medium diffusion) however much they may vary in other parts, this effect is something more than paralysis of the extremities of the nerves could cause. Paralysis of the extremities of the nerves would [nay], *must* lead to diffusion unless the cells themselves were affected. Then I think we need have no doubt whatever that the *cells*, independently of the extremities of the nerves, are affected in such a way that they no longer act as usual in obedience to stimuli, in other words that the cells have the functions of concentration and diffusion *paralysed*.

What I should like myself, therefore, would be to retain the expression "paralysis of concentration and diffusion", but to avoid the use of the expression paralysis of the concentrating and diffusing forces, or of the attractive and repulsive forces, as that implies what is incorrect regarding the physiology of concentration and diffusion, by giving the idea that during concentration the granules are all being attracted at once towards the centre, *and* vice versa.

One more point and one only: viz., regarding the publication of my supplement on the influence of the nerves on the arteries.

Mr. Goodsir has shown me a reference to Pflüger's[8] experiments on the large esculent frog, in which he found that division of the anterior roots of the spinal nerves causes relaxation of the vessels and stimulation of the same roots with galvanism causes the arteries to contract.

These are, I suppose, the experiments to which you had intended to refer me, but which I failed to find the account of. Had I been aware of them I dare say my experiments on this subject would never have been made; so that here, as in several other matters in this investigation, I have found myself anticipated. Yet at the same time I cannot but think some of my experiments deserving of publication, and it appears Mr. Paget thinks the same, but wishes me to make my results more precise. Now Mr. Goodsir agrees with me that experiments of the kind to which Mr. Paget alludes, viz., division of one half of the cord would be almost, if not altogether impracticable in the frog of this country. But I was aware of the possibility of making further observations regarding the seat of the nervous centre and have accordingly lately made some. In my paper I gave proof that a part of the cord as far forward as midway between the head and the caudal extremity does act as [a] nervous centre for the arteries after removal of the posterior half of the cord. What I felt might be further determined, but what I had not time to inquire into before the paper was read, was whether the posterior end in [the] tip of the cord is able to act as [a] nervous centre for the arteries after removal of the brain and the rest of the cord. Now this point I have since settled in the affirmative; I have found that after removal of the brain and all the cord except the posterior *tenth* part (in length) the arteries after a while recover their contractile and varying power. Now this seems to me of importance, as it proves that the portion of the cord which Waller[9] and Budge[10] found to be the "ciliary" centre, etc., etc., the nervous centre for the dilatation of the pupil, and which they also described as the centre for the contraction of the arteries of the face, is not the *only* part of the cord which has this office, at least for the lower limb. Thus this experiment along with my former ones proves that the posterior half of the cord contains throughout its length parts which act as nervous centres for the arteries of the feet. And other experiments render it probable that the same function is possessed by a part somewhat further forward. Now I believe this is about as much as can be made out on this point from the frog. I have also several times verified lately an observation I had made before viz., that after removal of brain and cord the arteries remain *permanently* relaxed, while on the other hand the experiment I related in speaking of the pigment, viz., dividing all the soft parts visible in the thigh except the artery and vein, after which recovery of contractile power on the part of the arteries occurs if the cord is entire, shews very strikingly how very slender a nervous communication between the cord and the arteries suffices to enable the former to influence the latter.

Now I believe I am not likely ever to carry this investigation much further, nor do I believe much good could be gained by so doing, I mean in the frog of this country, and Mr. Goodsir thinks that it would be well for the results I have got to be published to avoid the chance of being anticipated. If, therefore, you approve, I would rewrite this supplement on the influence of the nerves on the arteries, introducing my additional matter, and somewhat curtailing the old experiments perhaps. But I do not think it would be necessary to alter it so much as to require it to be published as a separate paper at a later period. It might simply be stated

that some new matter had been introduced with this part, the new parts being specified.

I find that there is yet *one* point Mr. Paget suggests that the supplement in the anatomy and physiology of the pigmentary system should be published as a separate paper *before* the essay on inflammation. My chief objection to this is that the chief illustration, i.e., figure of the pigmentary affair illustrates also a part of the "essay on inflammation". Also the last section of the paper is written as if the pigment subject was to be treated of in a supplement and I should prefer its remaining as such, if it might be so. This, however, is of small moment.

When must the paper be ready for printing? I am intending to be in London at Christmas for a week, and if that time would not be too late, I should very much like to consult with you personally as to the best way of getting the illustrations drawn. I should think that for the pigment cells, which require much delicacy of execution, Tuffer West[11] would be as good a man as could be found.

My father seems to think that some of the sketches on inflammation almost require the same kind of simple *colouring* that W. Jones'[12] plates in the bats wing in the Philosophical Transactions have. Certainly the plates would tell their tale much better, and the colouring would be only a *very* simple matter, viz., red for congested capillaries, pale red for arteries and arterial capillaries, and pale purplish for veins.

I am really exceedingly ashamed of this letter. I only hope you will not quite give me up as a bore.

Agnes unites in kind regards to Miss. Colville[13] and yourself with

Yours very truly,

JOSEPH LISTER.

W. SHARPEY, Esqr., M.D., F.R.S., etc.

11, RUTLAND STREET,
EDINBURGH.
28. *Nov*: /57.

MY DEAR DR. SHARPEY,

You will be wondering what has come over me that I have not long since thanked you for and replied to your most kind and to me highly satisfactory letter. Nothing indeed could have been more welcome to me. The reason of my delay in answering it has been the pressure of my present engagements, which have hardly left me any available leisure, except at times when the mind was too much fagged for work of such importance. To proceed, then to business. I mean to act on your suggestions from first to last. The section "on the state of the tissues in inflammation" will, I believe, be very much improved by remodelling it on the plan you have suggested, viz: speaking first of the facts actually seen in the pigment cells, viz: the "arrestment" of concentration and diffusion and then discussing the question of the "paralysis". That such "paralysis" really is present can, I think, hardly be doubted when the nature of the various agents which agree in causing

the " arrestment " is considered : for, while they differ among themselves exceedingly widely, they all have the common property of tending to destroy a part of the living body if maintained long enough in action upon it. It so happens, also, that the pigmentary tissue is a very delicate one, and is very apt to experience *permanent* injury without concomitant sloughing of the rest of the web. Thus if mustard be long enough applied, I find that the pigment cells begin to lose their sharpness of definition and appear to become disorganised. Similar effects on the pigment may also be seen in parts where mechanical violence has operated severely ; and after some days the pigment in such parts becomes absorbed, and when once so removed is never replaced. There is one point on which you began to speak to me one evening at Millbank, but the conversation was in some way interrupted : viz. the change the blood experiences in the irritated part when its corpuscles become adhesive. You objected to my making use of such an expression as implied that the tissues in a state of health *exercise* an *active influence* over the blood in their vicinity, keeping the corpuscles from becoming adhesive. You said I might as well say that, because a man who was in good health when in a snug room grew rheumatic if exposed to the outside air or if holes were made in the walls, *therefore* the walls of the room *exercised* an *active influence* for good upon the man. I have often thought of that criticism and admit its applicability. But, at the same time, I must contend for activity *somewhere*, either in the blood or the tissues ; just as the *man* in the room is active though the *walls* are passive, so, if the tissues are merely negative in their operation in health, the blood must have a positively active power. The *facts* seem to be these. In blood withdrawn from the body, the corpuscles, both red and white, are more or less adhesive ; but within the vessels of a perfectly healthy part are quite free from adhesiveness. In the vessels of a part which has been acted on by some deleterious agent, (but short of actual disorganization or chemical decomposition) the corpuscles shew the same adhesiveness as when the blood is removed from the body. But this is not all. If the agent have acted so as to produce only a temporary effect on the tissues (as e.g. a powerful galvanic shock, which only temporarily " arrests " the changes of distribution of the pigment molecules) then as the tissues recover the blood also regains its natural healthy characters : the corpuscles previously viscid and closely packed, become non-adhesive again and are driven out of the vessels in which they had accumulated. Now I say this *recovery* on the part of the blood implies something active *somewhere*. We may either suppose that the *healthy* tissues exert a purely *negative* influence on the blood, which consequently does not suffer as it does when in contact with glass or any other dead matter, which must in such case be supposed to exert a disturbing influence leading to changes in molecular arrangement probably, an influence which would seem, very likely, synonymous with the " attraction of aggregation " which leads to crystalization, etc., etc.—or else we must suppose that glass, etc., has a negative influence and the healthy tissues an active one upon the blood. (I fear I am writing obscurely.) But supposing the former hypothesis be correct, and that blood in a healthy part has its corpuscles free from adhesiveness because *not* subjected to the action of disturbing forces such as are exerted on it by glass, sweet oil, atmospheric air, or paralysed tissues ; then, I say,

the fact that, as soon as the paralysed tissues recover, the corpuscles return to their original state of non-adhesiveness implies that the blood has an independent power of recovery from its injured condition, (compare the case of the rheumatic man) so soon as the tissues cease to exert the said disturbing influence common to dead matter, and resume their healthy negative condition. Now at first this seemed to me very unlikely: viz. that glass, sweet oil, the atmosphere, and paralysed tissues should be the *active* agents (for evil) and that the healthy tissues should be merely negative in their operation on the blood, but I have been inclined to look more favourably upon this view from considering the phenomena of *coagulation* of the blood. I think it quite needless to allude to the simple facts which, to my mind, prove clearly that no *chemical* theory yet given accounts for the coagulation of the blood: for I believe you are quite satisfied of the insufficiency of these theories. In some way or other the healthy tissues (which may remain healthy as *tissues* for some time after death) *do not* make the blood undergo the changes which lead to coagulation which ordinary dead matter (such as glass, air, etc., etc.) and inflamed tissues (paralysed more or less) *do* induce these changes in the blood. The principle I am stating is well illustrated by the speedy coagulation of the blood at rest in an aneurismal sac, in contact *not* with healthy tissues but with mere coagulated fibrine or inflamed tissue. The contrast presented by such a case with the length of time that blood will remain uncoagulated after extravasation among healthy tissues is very striking. Again the same thing is seen perhaps even more remarkably exemplified by the fibrinous masses that accumulate about calcareous atheromatous spicula projecting into the aorta or other large vessels, when the "dead" matter determines coagulation of fibrine notwithstanding the rapid movement of the blood (though to be sure only on the *lee* side of the projecting mass, when the blood is, I suppose, more or less quiescent). Now, such cases as these look to me very much as if the "dead" matter with its ordinary "attraction of aggregation" was the *active* agent, the healthy tissues having a merely *negative* influence on the blood. Also the bulk of the masses of blood which may remain long free from coagulation after death (e.g., that in the auricles of the heart) but which coagulate so soon as exposed to the influence of the external world, seems to me inconsistent with the idea of the tissues exerting an *active* influence on the mass of blood, and so preventing coagulation. Now, if the influence of the tissues is purely negative as regards prevention of coagulation, this is a strong argument in favour of their being also negative as regards prevention of adhesiveness of corpuscles. Hence I mean to avoid the expression to which you objected.

I am glad to learn that you have verified the observation of the intermitting flow in the tadpole's gills. I alluded to Spallanzani's[14] observation in a note to my paper [p. 221], but did not lay very great stress upon it until I should be able to learn whether there is any *recoil* observed between the pulses. Can you inform me on this point? In the case of the young frog under chloroform there is certainly no recoil, only absolute quiescence, between the pulses. The case of the tadpole's gill seemed to me to differ from the adult frog's web in this respect that, as I suppose, there is only one auricle in the young tadpole as in the fish, or at least the right auricle is not brought into action for the

gills, and therefore the ventricle has to overcome the resistance of two sets of capillaries, the branchial and systemic, when driving the blood into the aorta. Now, it seemed to me that the resiliency of the elastic aorta beyond the gills might give rise to a recoil in the branchial capillaries, in which case the observation, though it would shew abundantly the superiority of the force of the heart to any other forces that might be conceived in operation, would not *prove* that no such forces did exist as causes of movement. With regard to the adult frog with weakened heart I have some doubt whether sufficient care has been taken to discriminate between the interruptions of flow due purely to weakness of that organ and such interruptions due partly to an unhealthy state of the blood with somewhat adhesive corpuscles. For I know that, unless attention has been specially directed to the matter, a very decidedly abnormal state of the circulation as regards the excess of slowly moving corpuscles may pass unnoticed.

One point I would ask your advice about is how the section " on the influence of the nerves upon the arteries " had better appear ; whether as a supplement, as it now is, or as a separate paper. Mr. Paget says it should appear " if at all, as a separate paper ", and Mr. Goodsir seems to think it forms such a distinct subject that it might be well to have it separate. In this matter I will be guided entirely by you. This only I would say, that if it be a separate paper it should appear, like that in the " pigment business " and in the same volume of the transactions, and, if possible, immediately before the others, or intermediate between them. I feel, for my own part, very indifferent about this, though I quite agree now that the *pigmentary* supplement will be better as a separate paper.

And now to reply to your question as to what things cause *concentration* and what *diffusion* of the pigment. First I would observe that there is this marked difference between the occurrence of concentration and diffusion under the influence of local applications, viz., that concentration occurs not only in the spot acted on, but in a greater or less area round about : in this respect concentration agrees with the contraction of arteries which occurs *round about* a part irritated, and both are obviously *reflex* phenomena, effected through the medium of the nervous system. Diffusion on the other hand occurs, as induced by a local application, only in the precise spot on which the irritant acts, e.g., beneath a little piece of mustard put on part of the web, but not at all beyond the limits of the mustard. Hence this diffusion is clearly due in some way to direct action of the irritant upon the part of the web on which it lies, and is not a reflex phenomenon. To this statement you will say I mentioned a contradictory fact in my last letter, viz., the *secondary* diffusion that Wittich observed after applying oil of turpentine to a moderately dark frog. This *secondary* diffusion *is* doubtless a reflex phenomenon, and is comparable to the secondary relaxation of arteries that occurs round about an irritated part. But I have never witnessed this *secondary* diffusion and possibly never shall in *our* frog, whose skin is far less sensitive to direct irritation than is that of the tree frog on which Wittich operated. Thus, oil of turpentine produces no primary concentration when applied to our frog, and its only effect on the pigment is to produce " arrestment " of concentration and diffusion. So little sensitive, indeed, is our frog to direct *stimulation*,

that the only cases in which I have observed concentration to occur from such local irritation are, 1st in an area round about a part pinched, 2nd beside the edges of a freshly cut wound, 3rd partial concentration has occurred in one leg in consequence of irritation of the roots of its nerves. The general rule respecting the operation of local irritants is that they deprive the part of the power of varying the distribution of the pigment, *leaves it just as it was*, whether fully concentrated, fully diffused, or in an intermediate state. To this general rule I have hitherto met with only three exceptions, viz., mustard, croton oil, and cantharides, which *before* they produce arrestment, produce a variable degree of diffusion in the spot on which they act. The effect is not always seen, and will be best seen when the agent acts least rapidly. Thus croton oil and cantharides act *extremely* slowly (not for one or two *hours*) and you will I believe always see some diffusion with them. But with mustard it is quite a matter of uncertainty, and if you use essential oil of mustard you are sure *not* to see any diffusion. "Arrestment" (paralysis) then takes place at *once*. But whether any diffusion occur[s] or not with mustard the ultimate effect (by the time stagnation occurs) is arrestment *both of concentration and diffusion*. As to the explanation of this variable degree of diffusion produced in the first instance by mustard, I am inclined to attribute it to paralysis of the extremities of the nerves occurring before the cells are fully paralysed, and so permitting a certain amount of diffusion to occur as always appears to do in health when the nervous influence is removed. That the extremities of the nerves are paralysed by the action of mustard is proved by the fact that if I put a bit of mustard on my own finger for an hour or two the spot loses its power of sensation.

By the way can you tell me if recent discovery has thrown any light on rigor mortis? Does it appear that the extremities of the nerves are concerned in its production? I am anxious to know this, in order to comprehend if possible, the remarkable post mortem concentration of the pigment.

Do you approve of the terms *concentration* and *diffusion*? They seem to me better than some others that might be used, because they imply not merely that the molecules are aggregated or dispersed, but also that they are collected with the centre of its cell or dispersed *throughout* the ramifying branches.

Many thanks as regard the colouring of the plates. I little intended so long a scrawl. Please forgive it if possible, and believe me, with kind regards to Miss Colville,

Yours very truly,

JOSEPH LISTER.

Many thanks for your kind proposal that I should make your house my town quarters at Christmas. Agnes desires me to unite her kind remembrances with mine. J. L.

11, Rutland Street,
Edinburgh,
3rd Dec. /57.

My dear Dr. Sharpey,

Among other things contained in the lengthy production which I last sent you was a request for information regarding the causes of rigor mortis. I now write to beg you not to trouble yourself to write on that point, as I have this morning obtained the information from Meissner's Bericht.

I also want to mention to you another set of observations I have made, which, though very simple, seem to me valuable with reference to the "paralysis" questions; which is, you know, a very important one as regards the essential nature of inflammation. It occurs to me that the cilia of the frog's mouth must perhaps be affected by agents which produce the "arrestment" of the pigmentary changes. I remembered your experiments with cilia, and recollected that your results were chiefly of a negative character, but then you had employed morphia and some other narcotics which, when applied to the web, do *not* cause inflammatory changes. I turn[ed] to your article in Todd's Cyclopædia, and found my recollections were in the main correct. I therefore proceeded to try the following experiment. Having cut off a small portion of the margin of a frog's tongue and placed it on a plate of glass under the microscope with just enough water around it to allow the cilia fluid to play in, I brought a piece of lint dipped in chloroform within about an inch of the object, so that the vapour might play upon it, while my eye was kept over the microscope. Almost immediately the cilia grew languid in their action and many stopped, reminding me of the speedy arrest of the flow of blood which occurs in the web when a drop of chloroform is applied to it. I then at once removed the lint, and soon the cilia resumed their action. I repeated this experiment several times. Then I tried strong liquor ammoniæ, proceeding in the very same way, having previously found that the pungent vapour playing for a short time on the *web* causes temporary arrestment of the pigmentary changes. The result was just the same, viz., almost immediate cessation of ciliary action, but recovery, *complete* recovery, after removal of the ammonia. I got similar results, but taking a longer time to develope themselves, by placing a piece of moistened mustard on the plate *near* the bit of tongue but not in the same water. I had previously ascertained that the pungent vapour of mustard will cause the pigmentary "arrestment", and that such arrestment may be only temporary. I also tried strong acetic acid, which readily induces inflammation of the web, operating as with the chloroform, so that its vapour might act on the bit of tongue. I only made one experiment with that agent, and it happened that the cilia were *permanently* arrested, I have no doubt because I applied the acid rather incautiously. For I must mention that the only difficulty in the experiment consists in applying the agents so delicately as only to cause *temporary* arrestment of the ciliary motion. There is no difficulty in making the cilia stop, but they will not recover their function unless you proceed very cautiously. I also found that the actual cautery brought near enough and not too near to the bit of tongue made the ciliary action languid without absolutely arresting it. The only one of your results which appeared at

variance with what I should have expected was that of the ciliary action going on in water impregnated with carbonic acid, for I had found that "aerated water" which I am assured contains nothing but carbonic acid, causes arrestment of the pigmentary changes. But then it occurred to me that very probably the carbonic acid might arrest the ciliary action also if *long enough* applied, just as the pigmentary arrestment and development of inflammatory stagnation of the blood require a considerable time of the action of the carbonic acid. And so it turns out. I put four bits of mucous membrane from different parts of the mouth and œsophagus of a frog in a glass of cold water, and four corresponding bits in a bottle of aerated water, and left both for about two hours. At the end of that time I found ciliary action in every specimen from the plain water, and not a trace of such action in any of those from the aerated water, although the cilia were distinct enough. There was, however, a great disposition to desquamation of the epithelium cells in the latter (from the carbonic acid water). I have since repeated this experiment on another frog with the same result. So far then, as I have tried, any agent that causes pigmentary arrestment when applied to the web causes also ciliary arrestment, and that not destruction of tissue, but suspension of function. Now there can, I conceive, be no doubt that the cilia, or the cells which bear them, are truly *paralysed* in these experiments, i.e., unable for the time being to act, and it is also *perfectly* clear that the effect is independent of nervous or any other influence from the rest of the body of the creature, for, as everyone knows, and as I saw abundantly in these experiments, the cilia continued to act after cells have been separated from the mucous membrane, *unless* you act upon them with one of these deleterious agents. The effect then is clearly a *direct* effect of the irritant on this form of extra vascular tissue. Do you not think that these simple experiments very much serve as experimenta crucis as regards the *nature* of the arrestment which irritants produce on the tissues, viz., that they paralyse. You see they do not in any way clash with yours, but it so happens that my former observations led me to try a set of agents which you had not employed. I see you found aether arrest the ciliary action; and I do not doubt it would excite inflammation if applied to the web. Hoping you will excuse me for thus trespassing again on your attention, I remain, with kind regards to Miss Colville, in which Agnes[15] unites,

Yours very truly,

JOSEPH LISTER.

W. SHARPEY, Esqr., M.D., F.R.S., etc., etc.

11, RUTLAND STREET,
EDINBURGH,
28th June /58.

MY DEAR DR. SHARPEY,

I heard the other day from Mr. Stokes to whom you forwarded my last to you, and his reply was in all respects quite satisfactory. Many thanks to you for procuring me the information it contained.

And now I must needs trouble you with another question: viz., whether a paper written now could have its abstract appear in the next number of the "Proceedings". I remember noticing in the number which contained the abstract of my inflammation paper that abstracts were given of some papers received after the Royal Society had ceased to meet for the season, some indeed as late as August.

I have been lately making some experiments in the so-called "hemmungs nervous system", a subject in which I have felt much interest from its connection with the origin of inflammation through the medium of the nervous system, as when the lungs inflame through long continued coldness of the surface of the chest. Some of my experiments with tepid water on the frog's foot had shewn that in the case of the *arteries*, relaxation of the muscular fibres in consequence of the application of an irritant to the skin is the result of the nervous centre (the spinal cord or those parts of it which preside over the arterial contractions) being thrown into a state of inaction. The very same stimulus whose *mild* action led to *action* of the nervous centre (with consequent contraction of the vessels) produced inaction of the same nervous centre when applied in a more intense degree. This was *proved* by the fact that the dilatation which occurs after contraction as the results of the application of tepid water of pretty low temperature gives place to contraction on re-application of water of the same temperature. Now—if the secondary dilatation was the result of any *action* of the nervous apparatus in consequence of the stimulus that dilatation would have been increased, instead of giving place to contraction, in repetition of the same stimulus. It is therefore clear that this secondary dilatation is the result of inaction of the nervous apparatus concerned. But it is quite impossible to draw the line between the dilatation which succeeds to contraction in consequence of a mild irritant, and that dilatation which occurs immediately, or, if you choose to assume it, after a "momentary contraction," after a stronger irritant, or the same irritant more strongly applied. Hence these experiments with tepid water lead to the remarkable conclusion that—given an afferent nerve, a nervous centre, and a motor nerve presiding over the contraction of unstriped muscular fibres—a very mild stimulus applied to the afferent nerve may (e.g., in the case of the arteries) lead to *action* of the nervous centre followed by muscular contraction, but a somewhat stronger stimulus applied to the afferent nerve may cause *inaction* of the nervous centre, and consequent muscular relaxation.

Having got this analogy to proceed upon I could hardly doubt that the same thing occurred in the so-called "hemmung system", viz., that if a mild enough stimulus were applied to the nerves, the contractions of the intestines and action of the heart would be increased instead of arrested. In order to put my ideas to the proof of experiment, I employed a coil battery, acting so feebly as to be only just appreciable to the tongue placed between its poles, and I have found the result such as I anticipated.

1st, as regard the intestines. I found the best mode of proceeding was to remove the skin and two layers of muscles from the abdomen of a rabbit, leaving the peritoneum and one layer of muscles, which are quite transparent enough to enable you to see any movements of the intestines, without the complication that the action of the air upon them involves. I found that

when a coil battery with its poles attached to the 6th and 12th dorsal spinous processes was put in action in the excessively mild manner I have mentioned, the writhing movement of the intestines became invariably greatly aggravated as long as the galvanic currents were acting on the cord, but as soon as the circuit was broken the former condition returned. But I also found that if the rods of soft wire were put into the helix, the " hemmung " action came into play and the intestines became relaxed and motionless : yet the action of the battery was very feeble, not even *unpleasant* to the tongue, when the rods were in. I also found that mechanical irritation of the spinal cord with a fine needle in the " dorsal region " (the same kind of irritation which I had always seen followed by contraction of the frog's arteries) produced increased contractions of the intestines. This I find from a paper to which Mr. Goodsir has referred me in Henle and Pfeufer's Zeitschrift for last year, has been observed also by a German, Spiegelberg[16] of Göttingen ; and his observations are particularly satisfactory as they were only incidental to the subject of his enquiry, viz., the movements of the uterus, and made before he heard of Pflüger's " hemmung " experiment.

I have also found, and this is so far as I know a new observation, that the increased peristaltic action which comes on after death in consequence of the cessation of circulation, stops when the " hemmung " action of the battery is set on. If you wait many minutes after death you will not see this, as the spinal cord soon loses its vital powers. But an experiment of the same kind may be repeated almost as often as you please in the following way. Tie with a fine needle and thread two or three principal arteries in the mesentery leading to about four inches of the intestine of a rabbit (the intestine lying upon a plate covered with fresh olive oil) and very speedily the movements, analogous to the post mortem movements, occur in this part and nowhere else. Then set on the strong action of the battery, whose poles have been previously attached to the spinous processes, and immediately the movement ceases. This experiment may be several times repeated. Of course it is necessary to tie the little arteries very delicately so as to avoid as much as possible the little nervous filaments. This experiment is of double significance ; first it proves very clearly that the increased peristaltic action that ensues on death is due simply to arrest of circulation in the part, for the " hemmung," on action of the battery, proves that the nerves have not been injured and therefore that the increased peristaltic action was not the result of injury of the nerves. 2nd, it proves that the arrest of the circulation does not *necessarily* produce peristaltic action, but that this post mortem peristaltic action, like that which occurs during life, requires that the nervous apparatus *in* the intestines should be in working order. I say the nervous apparatus *in* the intestine, for it is of course well known that the post mortem peristaltic action goes on after the mesentery has been cut off close to the gut. The existence of such a nervous apparatus distinct from the contractility of the muscular fibres is quite evident from the observation of Pflüger, which I have confirmed, that during the " hemmung " which results from powerful galvanic action through the cord, the intestines contract *at* any part irritated, though the movement does not extend to other parts ; the muscular contractility is unaffected although the co-ordinating apparatus is thrown

out of action. I have added further proof of the distinctness of these two functions, muscular contractility and co-ordination of movement, in the observation that the faculty of contracting *locally* on irritation remains in the intestines of a dead animal for some time after the power of peristaltic action has ceased: the nervous apparatus dying before the muscular. Mr. Goodsir has, since these observations were made, pointed out to me Meissner's[17] discovery of ganglia in the submucous tissue of the intestines, recorded in the last part of the former series of Henle and Pfeufer's Zeitschrift (1857); shewing the mechanism by which, no doubt, this co-ordination is effected.

Again I have shewn that the ordinary peristaltic action which occurs during the life of the animal is affected by the apparatus within the intestine: for if you cut in the mesentery the fine nervous branches leading to a certain portion, say three inches, of the intestine, taking care not to injure the blood-vessels you find that the peristaltic action goes on as in adjoining parts quite unaffected by the operation, whereas, if the peristaltic action was produced through the influence of the solar plexus or other nervous centres distant from the intestine, it should cease when the nerves in the mesentery are divided. Then, to prove that the nerves leading to the piece of intestine have been thoroughly divided, you have only to set on the strong galvanic action through the cord, when you see perfect rest and relaxation of all parts except that to which the nerves have been cut, and that continues to move just as before. This is a very striking experiment, and further proves that the nerves leading from the cord to the intestine must be in a state of *action* as far as the intestine in order to produce the "hemmung". From these and other observations with which I need not bore you at present, it follows quite clearly that the "hemmung" of the movement of the intestines is produced by the action of the nerves leading to the intestines upon the nervous centres contained in the gut, but that this is not the only effect of the action of these nerves, a milder action of the same nerves increasing instead of preventing the action of the co-ordinating nervous apparatus of the intestine.

Next as regards the heart, I find that if after dissecting the vagus of one side from surrounding connections without injuring it by pinching or otherwise, and isolating it by a bit of glass placed between it and surrounding tissues, you apply to it the poles of the coil battery tipped with fine silver wire, the battery acting so feebly as scarcely to be appreciable to the tip of your tongue, the beats of the heart are increased in frequency instead of diminished, but that if the rods of the soft wire are put into the helix the action of the heart becomes diminished in frequency, gets irregular and at length stops. This shews, as in the case of the intestines, how very gentle a stimulus must be used in order to produce *increase* of action of the ganglia presiding over the action of the viscus. This, however, is quite consistent with the great effect produced by slight stimuli in many other cases: e.g., when a touch of the skin at one point induces horripilation over a large extent of surface. But had I not been led to do so by the analogy of the arteries, before alluded to, I should probably never have dreamed of using a battery so excessively feeble in action. I see that here too I have been anticipated; for Spiegelberg alludes to experiments by Schiff[18] recorded in Vierordt's archives (which I have not been able to get) by which he is said to have shewn that a

gentle stimulus applied to the vagus promotes, instead of arresting the action of the heart.

One experiment mentioned by Pflüger in his book on the "hemmung nerven system" (1857) seemed strongly in favour of a constantly operating controlling action on the part of the vagus over the heart's contractions; viz., That division of the vagi increased the frequency of the heart's beats: even doubled their frequency. At the same time it is said that this has not been seen in *frogs*, only in mammalia. On this subject I made the following experiment. The abdomen of a rabbit having been for ¾ of an hour open and the intestines out upon a plate covered with sweet oil, the beats of the heart not having been as yet at all affected as far as I could judge, but being about 42 in 10 seconds, I exposed both vagi in the neck, taking care not to pinch them, but isolating them so thoroughly from the sympathetic that I could at any time divide them with facility without injury to surrounding parts. Having thus counted the pulsations in a coiling artery of the mesentery and found them 42, I rapidly divided both vagi in the neck, each with a momentary act with a pair of fine scissors, and about a minute and a half after the operation, found the beats 40 in ten seconds. A minute or two later the beats were 38, and so on, the pulsations gradually diminishing till ¾ of an hour after the operation they were 28. When they had fallen to 32 I pinched one vagus with forceps, and within about a minute of the commencement of the pinching, and while the irritation was still continued, the pulsations were again 40 in the 10 seconds. Similar results followed from the irritation on subsequent trials, but in a less marked degree. Finally after the chest had been opened and respiration had ceased, I found that the same irritation which had before increased the heart's action, viz., pinching the vagus, diminished the number of beats: the ganglia of the heart being, I suppose, so feeble as to be thrown into diminished action by the same stimulus which had previously induced them to act more.

In this experiment the fallacies which may arise from opening the chest were avoided, and it proves of itself quite conclusively that there is not *always* in operation a controlling action of the vagus over the heart's action. On the other hand I have observed in the case of the intestines that *invariably* during a prolonged struggle of the animal the peristaltic actions cease; which seems to show that natural and normal actions of the nervous system *may* induce the "hemmung".

If you can find time to set my mind at rest as to whether I shall prepare this paper for the Royal Society or do it in a shabby way for the *Lancet* you would do me a very great kindness. Of course in a case of this kind, where so many persons are working at the subject, it would be very desirable to have a notice of this paper early: and the proceedings of the Royal Society would of course be the best medium.

I need scarcely say that if the paper does go to the Royal Society, what would please me best of all would be for it to be thought worthy of a place in the Transactions as well as the Proceedings.

Agnes joins in kind regards to Miss Colville and yourself with

Yours very truly,

JOSEPH LISTER.

WILLIAM SHARPEY, Esq., M.D., Sec. R.S., etc.

11, Rutland Street,
Edinburgh,
7th July /58.

My dear Dr. Sharpey,

I fear my last letter to you must have arrived at a time when you were very much occupied, or else that you were very sceptical as to the trustworthiness of my observations. I suspect that the latter was the truth; for I admit that, considering the difficulty of the subject, and the variety of statement by different observers hitherto, anyone who goes to work at the "hemmungs nervous system" with a preconceived idea to support may be justly suspected of being unconsciously influenced, like the table turners, by his prejudice, and made to see things as he wishes rather than as they are. But I have endeavoured to bear fully in mind this tendency of the human mind, both in this and many previous observations, and, I trust guarded against it. Since I wrote to you I have made some additional experiments, two of which I will mention, for the sake of making you give some credence to those which I before related.

(1) It has been stated that division of the vagi produces in mammalia increased action of the heart. This, if true, would be strongly in favour of a constantly operating controlling action on the part of the vagus. I found it stated in Pflüger's book, however, that this effect had not been observed in frogs: and this seemed to me to throw doubt upon the alleged fact in mammalia. Accordingly I tested it by experiment. In 4 rabbits and a calf I have divided both vagi; I need not mention the details of the precautions used to avoid fallacy but may simply state, that in no single case was increase of the number of beats the result of division either of one or both nerves. Hence, even on the unlikely supposition that these were exceptional cases, these facts prove that there is not any *constant* controlling, i.e., checking influence exercised by the vagus over the heart's action.

(2) I had observed accidentally in one case in which I had been experimenting on the intestines after Pflüger's method (the poles of the battery being fixed to the 12th and 6th dorsal vertebræ) the vagi also having been both divided, the heart's action having grown very feeble was greatly improved by the application of galvanism to the cord. This shewed that the spinal system could influence the heart's action *otherwise* than through the vagi; and the same thing was proved by the frequently observed increase of the heart's action when the animal struggled, in cases where both vagi had been divided. Now it occurred to me that, if my view were correct, viz., that the nervous centres in the heart, intestines, uterus and probably other internal organs, were liable to "hemmung" from a little increase of action of the nerves (leading from the cord) whose *mild* action made these nervous centres more active; then the same kind of effects ought to follow from the irritation of other nerves proceeding from the cord to the heart as from irritation of the vagus; viz., increase of action with excessively mild stimuli, and "hemmung" with stronger stimuli. To test this I fixed the silver wire poles of my battery, one to the 6th dorsal spinous process, and the other to about the 2nd or 3rd *cervical* spinous process, so as to transmit the currents through

that part of the cord which sends off branches, through the sympathetic, to the cardiac plexus. I then found that when the action of the battery was the feeblest that I could recognize with the tip of my tongue placed between the fine silver wire poles, the heart's beats were increased by the galvanism so long as it continued in operation the number of beats in 10 seconds being raised 2 or 3, time after time, but after the circuit was broken and the action of the galvanism discontinued, the heart's beats fell to 5 or 6 beats below what they were before the galvanism was employed, and then after about 2 minutes regained their former number, e.g., in one case the beats were 37 or 38 before the galvanism was applied, 40 to 42 during the minute or two that the galvanism acted, then after it had ceased fell gradually but rapidly to 33, and then rose again to 37. These effects were observed time after time without exception; the increase in the beats during the galvanism being most marked at the commencement of the experiments, but the fall immediately after the cessation of the currents being as constant as it was striking. I could not but wonder that a degree of action of the battery so very faint should produce such effects. Then, when the rods of soft wire were put into the helix, or a little more acid into the jar, the action of the battery being still quite mild as felt by the tongue, the effect of connecting the circuit was to produce "hemmung", i.e., depression of the number of beats by about half a dozen *during* the action of the galvanism, and this time after time. I have experimented on two rabbits in this way, and in both found the results clear and satisfactory for a long time (about 2 hours), after which the galvanism failed to produce either increase or diminution of action of the heart; whether it was applied feebly as before or so powerfully as to elicit sparks from the parts of the tissues touched by the poles. It was quite evident that at these times the nerves concerned in conducting the stimuli to the cardiac ganglia were exhausted: and anyone repeating such experiments must be very careful not to use a strong action of the battery at first, or the exhaustion may occur very speedily, and then no effects upon the heart will shew themselves. I should mention that the counting of the beats of the heart with precision was greatly facilitated in my last experiment (done yesterday) by removing a portion of skin and pectoral muscle from the præcordial region of a half-grown rabbit, when the heart's action could be seen with the utmost ease through the transparent intercostal muscles and pericardium.

So this experiment confirms the general doctrine of the liability to diminished action of the part of the nervous centres seated in the viscera, in consequence of increase of the action of the nerves leading from the cord to them: and further shews how extremely delicate must be the stimulus applied to the cord or the nerves in order that the *increased* action of the nervous centres aforesaid may be developed. Also it shews how liable the nerves leading from the cord to these nervous centres are to lose their function in consequence of long continuance or excess of the stimulation. I expect to be in London at the end of this month, and, if you thought fit, would bring with me an abstract of a paper on this subject: for I really think I have now got facts enough to warrant me in publishing. If you do not approve of this plan, I would send a paper on the subject to the *Edinburgh Medical Journal*

for next month. The paper would have to be differently got up according to the medium in which it was to appear, so there seems no time to lose. May I then ask the favour of an early reply.

Yours very truly,

JOSEPH LISTER.

WM. SHARPEY, Esq., M.D., Sec. R.S.

11, RUTLAND STREET,
EDINBURGH,
28th Nov. /58.

MY DEAR DR. SHARPEY,

I am much obliged to you for the information conveyed in your kind note received this morning. I dare say my third (and last) paper might have been at the printer's before now, had not a recent letter from Tuffer West made me give up hope of getting it into the next part of the Transactions. Now, however, you may be sure, I will not waste a minute, for I am very desirous to get the job off my hands. Half the last paper is already fit for the press, and I will send it at once, and get the rest done in a few days.

One advantage this delay has had; viz., it has shewn that I was wrong, and you were right after all about the matter of the coagulation of the blood. Your view was that the reason of coagulation on removal from the vessels or on injury or disease of the vessels was a difference of *surface* in the surrounding objects. Mine had been that surrounding objects were neutral as regards the blood, but that the tissues of the vessels were active in preventing any tendency to escape of ammonia, etc., etc. My reason for this view was that I found that air freely admitted into the vessels along with the blood had no effect in causing coagulation. Hence, as exposure to air has been supposed by so many physiologists to have great effect in causing coagulation, I rashly inferred that because the atmosphere was neutral in its effect on blood within the vessels, therefore *all* dead matter was so, *solid* as well as gaseous. But by some recent simple experiments upon sheep's feet I have found that if a piece of fine silver wire, wax, hair, glass, etc., etc., be introduced into a vein, the fibrine deposits upon the solid and, if the mass be considerable, the clot may be large; although mere surrounding of a vein without introducing any extraneous solid does not cause coagulation except in the mouth of the wounded vessel. From these and other facts, which I must not detain you now with detailing, it seems to follow that the lining membrane of the vessels (and probably other tissues also) differs from any ordinary solid substances I have yet tried in being *neutral* as regards the tendency to cause deposition of the fibrine from solution, while silver, wax, etc., and the tissues themselves when diseased, attract the fibrine or in some way determine its precipitation, probably as a crystal does a solid in solution. Outside the body it appears that a certain small amount of ammonia is necessary to keep the blood fluid, but within the vessels the ammonia seems not at all essential to the maintenance of the fibrine in solution. For it is easy to prove that most of the ammonia, if not all of it, escapes from the

blood in the vessels of an amputated sheep's foot, when the foot has been kept a few hours or when a vein has been wounded. Under these circumstances the blood let out from a vessel coagulates immediately, so that I have seen evidence of the commencement of the process within 5 seconds of the shedding of the blood. This rapid coagulation must I imagine be attributed to escape of ammonia; yet if the blood thus devoid of ammonia or nearly so be left in the wounded vessels it will remain fluid for any length of time, hours or days.

I have been induced to write you a much longer account of this matter than I intended, but I hope you will excuse this. Mr. Syme has returned safe and sound, and apparently well pleased with the results of the council meeting. He gives a good account of your health.

Agnes unites in kind regards to Miss Colville and yourself with

Yours very sincerely,

JOSEPH LISTER.

Excuse great haste.

WILLM. SHARPEY, Esqr., M.D., Sec. R.S., etc.

(To be continued.)

SHORT NOTES OF RARE OR OBSCURE CASES

A TUMOUR OF THE MOLAR GLANDS.

By C. GRANTHAM-HILL,
SENIOR SURGEON, KHARTOUM HOSPITAL.

The patient, a Sudanese woman of about 50 years of age, complained of a large and painful swelling on the left side of the face (*Figs.* 61, 62). She noticed it first as a small swelling near the left angle of the mouth a year previously, since when it had steadily increased in size, and had, during the last two months, become painful. On palpation the tumour was lobulated and elastic and was not attached to the jaws or parotid gland, being freely movable. It was thought to be a sebaceous adenoma.

Operation.—After preliminary removal of some carious teeth and cleaning of the mouth, the inferior dental nerve was blocked with novocain inside the mouth, and the growth surrounded by a subcutaneous infiltration block of the same solution. The growth was easily dissected out, following a well-defined capsule, which was not adherent to the surrounding tissues, excepting to a small area of buccal mucous membrane near the opening of Stenson's duct. This piece of mucous membrane was removed along with the tumour without interfering with the duct. Enough skin was saved to cover the wound. Post-operative recovery was uneventful except for a small fistula which formed at the point at which the mucous membrane had been removed, and which closed spontaneously. (*Fig.* 63.)

Pathology.—I am indebted to Dr. T. F. R. Hewer of the Welcome Tropical Research Laboratory for the following report :—

"The tumour weighs 530 grm. and measures roughly 12 by 11 by 8 cm. It is a coarsely lobulated mass whose borders are well defined and encapsulated : the overlying skin is thinned over some of the larger bosses of the tumour, but even here there is no invasion beyond the capsule, and the skin can readily be stripped off. The tumour is firm and resilient, and on section it offers considerable resistance to the knife. The cut surface, after fixation, shows irregular trabeculæ of dense connective tissue, partly hyaline and partly opaque, passing from the capsule to the centre of the tumour mass. The parenchyma consists of small irregular lobules of pinkish grey or white colour, lying within a fine meshwork of connective tissue. At the centre of the tumour is an area of hæmorrhage in which the parenchyma has been replaced by masses of soft friable colloid and blood-clot ; nearer the periphery there are also a few chalky or caseous patches of necrosis. There does not appear to be any deposit of calcium, nor is there any sign of cyst formation.

"Microscopical examination of a section reveals a solid structure of large polyhedral epithelial cells arranged in lobules circumscribed by fine septa of connective tissue; these cells have a granular cytoplasm and a large dark-

Figs. 61, 62.—Tumour of the molar glands.

Fig. 63.—Appearance of patient after operation.

staining nucleus. There are occasional areas of necrosis in the centres of the lobules, and, in other parts, clefts have arisen which give an appearance of acini, but in reality there is no suggestion of such a formation. All parts of

the section contain the same type of cell; there is no sign of duct formation or of any other tissue. The tumour would seem to be a solid adenoma of a simple salivary gland." (*Fig.* 64.)

Comment.—There is reason to believe the tumour to be one arising from the molar glands, described in *Gray's Anatomy*[1] as being, " four or five, placed between the masseter and the buccinator muscles around the distal end of the parotid duct ". C. E. Shattock[2] (1929) says that these may give rise to rare tumours similar to mixed parotid tumours, but in front of, and quite separate

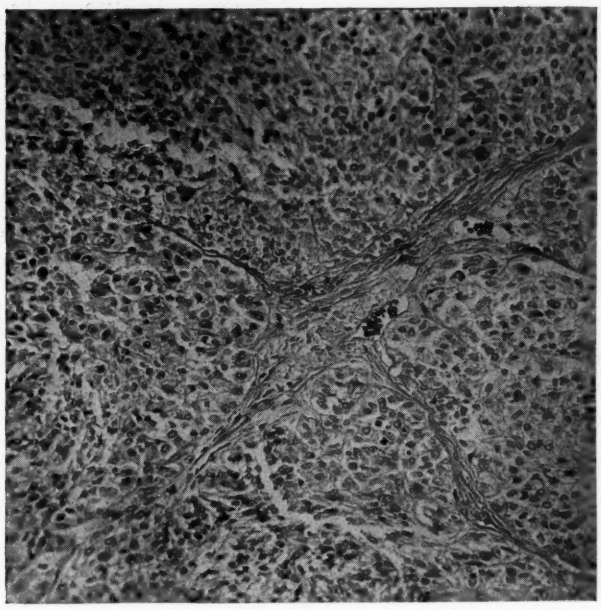

Fig. 64.—Microscopical appearance of tumour. (× 100.)

from, the socia parotidis; that the tumour may spread through the capsule and infiltrate the surrounding tissues, though at first movable or rounded or lobulated; and that it may extend forwards almost to the angle of the mouth. Lenormant, Duval, and Cottard[3] (1908) collected ten cases of tumours of this type, some occurring on the lips. Some contained bone and cartilage, some were cystic, and some solid. None were malignant, and nearly all occurred in young people.

I am indebted to Dr. H. M. Elliott for sending me the case; to the Director, Sudan Medical Service, for allowing me to publish it; and, particularly, to Professor Arnold K. Henry, of Cairo, for references in the literature.

REFERENCES.

[1] *Gray's Anatomy*, 1918 ed., 1046.
[2] SHATTOCK, C. E., *Handbook of Surgical Diagnosis*, 1929, 461.
[3] LENORMANT, DUVAL, and COTTARD, " Les Tumeurs mixtes de la Joue et des Lèvres ", *Rev. de Chir.*, 1908, xviii, 1.

THREE ENTEROLITHS IN A SINGLE COIL OF JEJUNUM.

By R. E. KELLY, C.B.,

SENIOR SURGEON, LIVERPOOL ROYAL INFIRMARY.

Stercoliths, although common in domestic animals, are somewhat rare in man. They are generally single, may attain the size of a golf ball, and usually cause intestinal obstruction in the small bowel. A rounded impediment of this kind may easily be mistaken for a gall-stone, from which, indeed, it can only be distinguished by chemical analysis.

Their cause is obscure. The present case would seem to show that they may be precipitated in a dilated and hypertrophied coil of intestine proximal to a stricture. Further, the case is interesting as showing three enteroliths all lying in a single coil of jejunum, and all imprisoned between two strictures in the gut so that they could pass neither onwards nor backwards. The result was that by their ball-valve action on the distal stricture this particular coil of jejunum had become so dilated and hypertrophied that it resembled a second stomach. The coil was about 10 in. long by 2 in. wide.

The three enteroliths were of varying sizes, so that it is probable that they were not all formed at the same time. Perhaps the damming back of the intestinal contents by one enterolith favoured the deposition of the other two. In this patient their formation probably took many years.

History.—Twenty years ago I operated on a girl of 12 for an 'acute abdomen'. The diagnosis had been appendicitis. This was a mistake, for the appendix was normal. There was a suppurative pelvic peritonitis due to a perforation of a tuberculous ulcer in the jejunum. The perforation was stitched up and the pelvis drained. She made an uninterrupted recovery and for ten years was quite well.

For the past ten years she has had attacks of vague abdominal pain. The pain bore no relation to food, had a sudden onset and sudden cessation, was generally centred about the umbilicus, and usually lasted for several minutes. She described it as being colicky in character, and though it was occasionally felt in the back, it did not radiate into either the shoulder or the groin. Vomiting occurred when the attacks of pain were especially severe. The vomit was never excessive, and sometimes contained bile. The appetite was good, and she did not avoid fats. She has taken aperients for years, but did not think that the pain was worse when she was constipated. During the last four months the attacks have been much more frequent and severe. There has been no loss in weight.

On Examination.—The patient was admitted to the Royal Infirmary, Liverpool, and a complete X-ray examination was made by Dr. Roberts. He reported that there was a dilated coil of small intestine showing signs of obstruction. The rest of the alimentary tract was normal. From the radiograph the dilated coil seemed to be near the ileocæcal valve, but this appearance was false. It was pelvic in position, but was 6 ft. from the duodenojejunal juncture. The dilated coil was about three times the diameter of the proximal jejunum and at least four times the diameter of the succeeding

bowel. The whole coil was some 10 in. long, and there was a stricture of the bowel at either end of the dilatation, the distal stricture being much narrower than the proximal stricture. The three stercoliths were prominent

FIG. 65.—Inside of dilated coil of jejunum with three enteroliths *in situ*. A, Distal bowel; B, Stricture; C, Stricture; D, Proximal bowel.

FIG. 66.—Outside of jejunum. A, Proximal bowel; B, Distal bowel.

features even from the outside, and they could not be moved from the dilated coil. (*Figs.* 65, 66.)

Operation.—This consisted of resection of the coil and its contained concretions. The bowel was restored by closure of both ends and a side-to-side anastomosis. The convalescence was uninterrupted.

Pathological Report.—Dr. Howel Evans reports on the concretions as follows :—

"The largest concretion is irregularly pyramidal in shape, the sides measuring approximately 2×5 cm. The second is flat and triangular, $2 \times 2 \times 0{\cdot}5$ cm. The third is similar in shape to the second but somewhat smaller. They are all smooth and polished like pebble stones. They show a thick smooth brown outer layer, and the concretion which was examined could be crushed between the fingers, showing a loose friable centre. The material is largely organic, probably fatty in nature as it is readily soluble in alcohol, leaving a greasy stain when the alcohol is evaporated. The brown colour is chiefly due to urobilin. Tests for unaltered bile-pigments are negative. On extraction with chloroform, cholesterol cannot be demonstrated either by crystallization or chemical tests. Inorganic matter is scanty. Traces of calcium are present, but phosphorus, a frequent constituent of intestinal concretions, cannot be found. Assuming that the two other concretions are similar chemically as they appear to the naked eye, the evidence is against gall-stones and in favour of organic material, largely fats and soaps, which have been formed *in situ*."

A complete analysis of two enteroliths is given in a paper by James Phillips, of Bradford.[1]

REFERENCE.

[1] Phillips, J., *Brit. Jour. Surg.*, 1920–21, viii, 378.

GASTRO-INTESTINAL CRISIS OF ANGIONEUROTIC ŒDEMA.

By R. J. McNEILL LOVE,

SURGEON, ROYAL NORTHERN HOSPITAL, LONDON.

Abdominal symptoms of angioneurotic œdema have been observed on numerous occasions. Sir William Osler[1] describes a case of a man, age 58 years, who had been affected since childhood with attacks of abdominal colic, retching, and vomiting, two months being the longest interval of freedom. These attacks were associated with patches of œdema, but never of purpura. In the same paper an account is given of a woman of 45 who for five years suffered from attacks of urticaria and œdema of the hands and lips. These manifestations were associated with periods of vomiting which lasted from ten to twenty-four hours. Grey powder and calcium lactate resulted in complete cure. More recently a fatal case of Quincke's œdema with painful abdominal crises has been described.[2]

The case described below is of interest in that radiological abnormalities were evident during the gastric crises, and caused some discussion as to their correct interpretation.

Miss P. B., age 31, stated that she had always been healthy until two years ago. One Saturday evening, while dressing in preparation for a dance,

she found to her surprise that she could see her upper lip. The lip became rapidly swollen, and she was unable to attend the dance, and went to bed instead. On the following day she suffered from abdominal discomfort, but the lip improved, and she returned to work the day after. Since then she has been subjected to sudden œdematous swellings, chiefly of the face, lips, and forearms, and attacks of violent abdominal pain and vomiting. These

FIG. 67.—Radiograph showing peculiar condition of pyloric portion of stomach, presumably due to œdema.

attacks are ushered in with a feeling of constriction affecting the throat and dysphagia.

A fortnight seldom passes without her being confined to bed for one or two days. Recently these attacks were so severe that she was admitted to the Metropolitan Hospital under Dr. J. W. Linnell. A bismuth meal (*Fig.* 67) revealed a curious condition which was considered to be possibly a polyposis of the pylorus, although the diagnosis of an angioneurotic œdema was held to be more likely. However, as the abdominal symptoms had increased in

severity, and the patient yearned for any possible relief, a laparotomy was performed four days after the radiograph had been taken, in order to exclude any permanent pathological condition. The stomach was practically normal except that the pyloric portion was slightly more red and glistening than usual, and the individual muscular bundles were more apparent. The abdomen was closed, and six days later a radiograph (*Fig.* 68) revealed no abnormality.

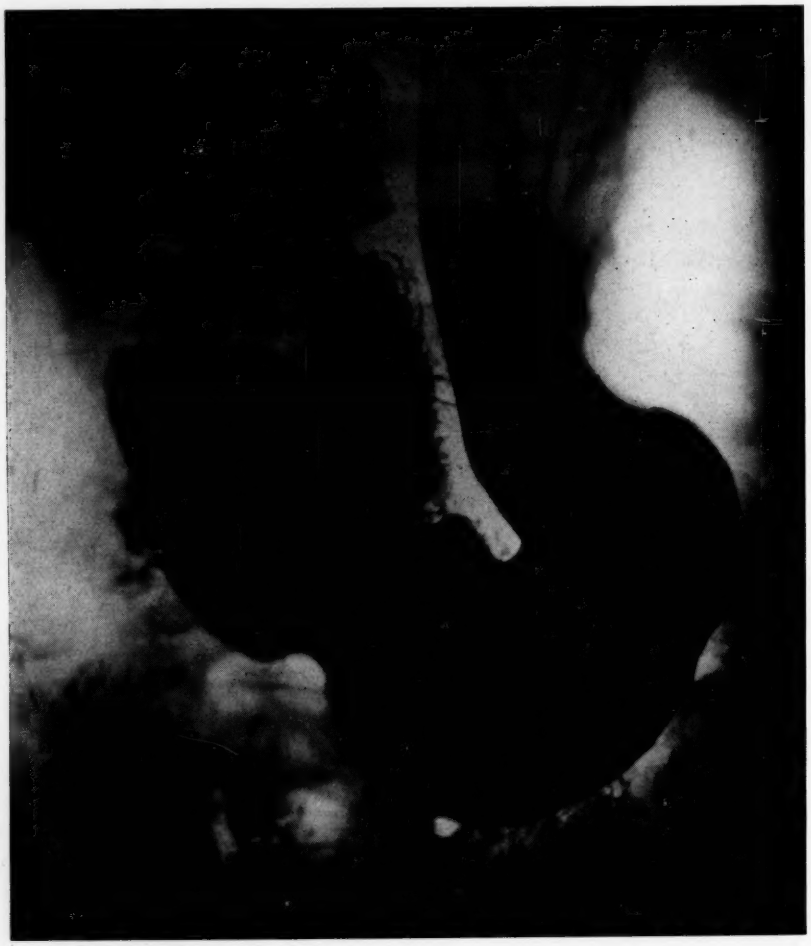

FIG. 68.—Radiograph taken ten days later, the stomach now being almost normal.

Three months later (Jan. 12, 1932) the patient reported that although she had had further attacks they were " not so bad and less often ".

As is usual in cases of angioneurotic œdema, the disease in this instance was hereditary. The patient's father and one of his brothers both suffered from the more common subcutaneous manifestations.

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[1] OSLER, SIR WILLIAM, *Brit. Med. Jour.*, 1914, No. 1.
[2] VALLERY-RADOT, *Bull. Soc. méd. Hôp. de Paris*, 1931, March 30.

REVIEWS AND NOTICES OF BOOKS.

Tumours of the Breast: their Pathology, Symptoms, Diagnosis and Treatment. By Sir G. LENTHAL CHEATLE, K.C.B., C.V.O., F.R.C.S., Consulting Surgeon and Emeritus Lecturer on Surgery, King's College Hospital, London; and MAX CUTLER, B.Sc., M.D., Director of Tumor Clinic, Michael Reese Hospital, Chicago. Large 8vo. Pp. 596 + viii, with 468 illustrations and 18 coloured plates. 1931. London: Edward Arnold & Co. 50s. net.

THIS book is the outcome of thirty-five years' study of the breast, and is based on the examination of whole microscopical sections in series which Sir Lenthal Cheatle has developed to such a high degree of perfection. All pathologists and surgeons intimately concerned in the study and treatment of diseases of the breast must have read with interest the numerous papers Sir Lenthal Cheatle has written on these subjects. This volume, in which he has gathered his accumulated material and expanded his theories, will be very welcome to them—none the less because some of the matter it contains is controversial.

The first chapters are devoted to a careful description of the anatomy and physiology of the breast, and the authors have collected in an attractive form all the available knowledge on these subjects. After a survey of the congenital and acquired anomalies of the breast they discuss the natural laws in pathological growth and the relation between hyperplasia and neoplasia.

Perhaps the most important part of the book is that formed by the chapters dealing with the medley of conditions at present included under the heading of 'chronic mastitis'. The authors believe chronic mastitis to be much more a physiological process than a pathological one, and a process of inflammation least of all. To the condition they apply the term 'mazoplasia' and consider it has no relation to carcinoma production. From this they clearly differentiate the condition they call 'cystophorous epithelial hyperplasia', which is one of pathological activity and can be traced in all stages of transition to the neoplastic type, which may consist either of benign or malignant tumours. About 20 per cent of all carcinomas of the breast can be directly traced to this method of origin. They propose to limit the term 'chronic mastitis' to those conditions where definite evidence of the existence of inflammatory changes is forthcoming.

The evidence in support of the authors' suggestions is clearly stated and illustrated. Many observers will, however, feel that the problems of chronic mastitis are too complex to be solved in this simple way, and will therefore prefer to retain the admittedly unsatisfactory term of 'chronic mastitis' until they are convinced that these new terms are not leading to worse confusion. A more authoritative criticism of these views is to be found in the Bradshaw Lecture on chronic mastitis by Sampson Handley (*Lancet*, 1931, Nov. 28).

The sections on cancer of the breast throw new light on the subject, for by their whole-section method the authors have separated a large number of anatomical types not generally differentiated in published series. They do not favour histological grading as a method of prognosis, and indeed it is clear that this method can only be taken into consideration within each distinct anatomical group. As regards the treatment of cancer of the breast, the authors favour radical operation followed by external radiation. Radiation alone is used on those patients who refuse operation or who are unsuitable subjects for operation.

In the chapter on Paget's disease, the various views of its etiology are outlined and an interesting and detailed study of seventeen cases is used as a basis for the interpretation advanced.

In the last chapters, which are devoted to a review of radiation treatment, the action of radium and the factors in radio-resistance are discussed in relation to breast tumours, and the various current views on the efficacy of radiation treatment of cancer of the breast in all stages, and of its metastases, are impartially given. There is much information in these chapters that is indispensable to the general practitioner, to whom patients must always turn for sound advice as to whether radium or surgery is to be used. The book should therefore appeal, not only to the scientific worker, but also to a wider circle of clinicians with whom the ultimate settling of pathological details ranks low in importance.

The volume is arranged so as to be used as a work of reference and as an atlas. It is lavishly provided with illustrations, and both authors and publisher are to be congratulated on the way in which it is presented.

Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London. Third edition, revised and enlarged. Medium 8vo. Pp. 277 + xx, with 318 illustrations, some of which are in colour. 1931. Bristol: John Wright & Sons Ltd. 21s. net.

THE first edition of this work was reviewed in the BRITISH JOURNAL OF SURGERY just after its appearance in 1927. It was then hailed as an admirable book, setting out in a pictorial way a series of demonstrations of the physical signs of clinical surgery, and it was confidently recommended to the notice of all students. Your reviewer can do nothing better than reiterate this notice, and say that the book has maintained the standard which was then set, and has now established itself as a work which every student ought to possess.

At the present time, when the accessory methods of diagnosis are so largely practised and when attempts are made to learn clinical surgery by easy routes, it is more than ever necessary that students should be reminded that nothing can ever take the place of close contact with actual clinical material, the study of which will supply the framework on which all other methods of diagnosis must be based. This book helps very largely to this end, not to supplant such clinical examples, but to assist the student to make the best use of the material which is presented to his notice.

This edition is more liberally illustrated than ever, but a good many of the pictures are really superfluous, and if by a judicious reduction of their number the price of the book could be lowered a little, the student would have every reason to be grateful.

Although the illustrations are nearly all worth framing, there are a few exceptions. For instance, *Fig.* 80, showing a mastoid abscess, should be replaced by a photograph of an actual case, and *Fig.* 86, of cavernous sinus thrombosis, is not sufficiently characteristic to convey the main features of that striking condition to the student. Surely it would not be difficult to get a better picture of a backward dislocation of the elbow than the one shown on page 99? *Figs.* 21 and 22 ought to be strictly comparable, and it would add greatly to their value if a picture of gummata on the leg could be substituted for those on the chest. It might be a help for future editions to point out that tumours in muscles, as well as moving with the muscle, are also fixed when the muscle is held in contraction. In dealing with the examination of the thyroid it is rather amusing to have the time-honoured sign associated with the act of swallowing attributed to such a comparative youngster as our friend Lahey. It also seems a pity to omit to mention the valuable aid which we often get from the use of X rays in the elucidation of these swellings.

Fig. 42, illustrating the method of determining the relation of a swelling to the sternomastoid, is a little misleading, for it certainly suggests that the patient is trying to force her head towards the affected side, though the text clearly explains that it should be in the opposite direction. In speaking of thyroglossal and branchial fistulæ, it would be well to mention that the secretion is characteristically sticky. As the text asserts, the risus sardonicus is supposed to resemble a smile, but the smile is surely a painful one, and this is borne out by the wonderful illustration borrowed from John Thomson. In the description of the tests for dislocation of

the shoulder, one wonders why those attributed to Callaway and Duga should be designated by their authors' names, while poor Hamilton is not given credit for his valuable straight-edge test.

In dealing with the examination of the liver for enlargement, it is a mistake to give the impression that inspection is of very little value. Quite often not only can the enlarged liver be seen, but the umbilication of the nodules may be perfectly obvious if the patient is well relaxed and in a good light; and quite apart from that, the general enlargement of the hepatic region is often very striking. Of course it is possible that the author assumes that such gross conditions will never present a problem in diagnosis, but a considerable clinical experience leads the reviewer to assert that such is unhappily fallacious. In dealing with the umbilicus it would have been well to mention the nodule of malignant growth which is often so helpful in elucidating a doubtful case.

One questions, in these days, whether Trendelenburg's test for varicosity of the internal saphenous is always an indication for the operation associated with his name. Rather it appears as though operations for varicose veins had been entirely superseded by the easier and more satisfactory method of injection.

But these are very minor points, and they do not detract much from what is undoubtedly a very useful and valuable book.

Die Schleimhaut des Verdauungskanals im Röntgenbild. By HENRI CHAOUL and ALBERT ADAM, with a Foreword by FERDINAND SAUERBRUCH. Imperial 8vo. Pp. 229 + viii, with 219 illustrations. 1931. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 22.50; bound, RM. 25.

THIS volume is a record of work in a field which has hitherto attracted more attention among Continental radiologists than in this country. The critical radiographic study of details of the alimentary mucosa which received its initial impulse from the work of Forsell, has been advanced by many workers. In this field Chaoul, himself a pioneer, has developed a technique and special apparatus which facilitate and lend precision to this somewhat laborious and time-consuming work, which consists essentially in radiographing the organs when outlined by *very small quantities* of opaque emulsion, under suitable compression.

A fundamental fact which emerges from the work under review is the constancy of the arrangement of the gastric mucosal pattern in the same individual, and to some extent in different individuals. This is at variance with the views of Forsell and of Berg, but the authors appear to have proved their case. They discuss fully the influence of tone and peristalsis on the mucosal pattern, and ascribe to these factors, rather than to independent contraction of the muscularis mucosæ (Forsell), the predominant rôle in producing its momentary variations.

In many respects their study of the normal is the most valuable part of the book. On the pathological side there is little which is new, though the standard of technical achievement compels admiration, and many excellently reproduced radiograms of small lesions, which might easily have escaped recognition by ordinary methods, prove the value of the authors' technique; for example, peptic ulcers of the œsophagus, early carcinomata of the stomach, gastrojejunal ulcers, and a striking case of papillomata of the colon.

We had hoped that their researches might throw new light upon the diagnosis of gastritis and colitis, and particularly upon the causation of 'mottling' of the mucosa which, in a radiogram, often suggests these conditions. These appearances are discussed, and such causative factors as mucosal hypertrophy and atrophy, papilliform swelling, and deposits of mucus are differentiated, but it is clear that the accurate radiological diagnosis of gastritis and colitis requires further study, both radiologically and pathologically: in the meantime we must endorse the rather cautious views taken by the authors towards these problems.

The book may be heartily recommended to gastro-enterologists and to radiologists, who will find it both informative and stimulating. There is a very complete bibliography.

Lehrbuch der Röntgendiagnostik. By H. R. SCHINZ, W. BAENSCH, and E. FRIEDL, with the collaboration of M. HOLZMANN, A. HOTZ, O. JÜNGLING, E. LIEBMANN, E. LOOSER, and K. ULRICH. Third edition. Two volumes. Imperial 8vo. Pp. 1623 + xxviii, with 2714 illustrations and 5 photographic plates. 1932. Leipzig: Georg Thieme. Paper covers, M. 214; bound, M. 220.

THIS comprehensive text-book of radiology has won a well-merited international reputation. The third edition represents a very distinct advance upon the earlier ones, both in the wealth of additional matter and in the elaboration of the original sections, which have been largely rewritten. One of the most valuable features of this book has been the authors' insistence upon the pathological basis of X-ray appearances: this aspect is even more fully emphasized in the new edition. For example, in the section on bone growth and diseases of bone are collected illustrations of normal and growing bones with clear descriptions of normal growth processes. Diseases of bone are dealt with on similar lines, with photographs of specimens, microphotographs, and key diagrams side by side with the radiograms. This section contains also descriptions of rare bone diseases and of conditions which have been only recently studied in detail by X rays. In particular the chapters on diseases of the vertebral column have been rewritten in the light of the revolutionary work of Schmorl.

In compiling this work the authors owe much to their collaborators (Holzmann, Hotz, Jüngling, Liebmann, Looser, and Ulrich). In addition, the best recent work in all countries has been laid under contribution, including that of the American registry of bone sarcoma; German work on tuberculosis of the lungs, Scandinavian and German work on the alimentary mucosa, and French work on cardiology. The chapter on radiology in gynæcology is new, and that dealing with urology has been brought up to date by inclusion of new methods in urography. The excellent articles by Jüngling on myelography and ventriculography remain as originally written.

About half the second volume is devoted to the chest, and half to the alimentary tract. The sections dealing with pulmonary tuberculosis and with radiology in connection with thoracic surgery are specially useful. The alimentary tract is extremely well done, with emphasis throughout upon 'direct' as opposed to indirect signs. A full index and really useful bibliographies—the latter placed where they are most useful, at the end of each short section—are good features.

Very high praise is due to the publishers for their share in this work; particularly for the radiograms, which reproduce the crispness and clarity of the originals. Their wide experience of this type of work, and their immense material, have enabled them to publish this book supremely well; and it is good to find collected within its covers material which has already appeared in periodical form, but which in that form is difficult of access. This book is the best and most complete modern text-book on diagnostic radiology that exists, and it should be in daily use by every radiologist and on the shelves of every important medical library.

Atlas de Radiographie osseuse.

I. **Squelette normal.** By G. HARET, A. DARIAUX, Electro-radiologistes des Hôpitaux de Paris; and JEAN QUÉNU, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. With the collaboration of H. P. CHATELLIER, Oto-rhino-laryngologiste des Hôpitaux. Second edition, revised and enlarged. Preface by PIERRE DUVAL. Large 4to. Pp. 186, with 149 illustrations. 1932. Paris: Masson et Cie. Fr. 200.

II. **Pathologie.** In two volumes. Vol. I. Lésions traumatiques. By G. HARET and A. DARIAUX, Electro-radiologistes des Hôpitaux de Paris; and JEAN QUÉNU, Professeur agrégé à la Faculté de Médecine, Chirurgien des Hôpitaux de Paris. Vol. II. Lésions non traumatiques. By ETIENNE SORREL, Chirurgien de l'Hôpital Trousseau; and Mme. Y. SORREL-DEJERINE, Ancien Interne des Hôpitaux. Large 4to. Pp. 344, with 897 illustrations. 1931. Paris: Masson et Cie. In two volumes, Fr. 310; in one volume, Fr. 280.

IT is difficult to do justice to this magnificent work, the first part of which has already entered upon a second edition. The first part gives a most complete radiographic presentation of the normal skeleton. First, the bones of an adult, each from

several different points of view. Second, the bones of a child at various ages from intra-uterine life up to puberty. Third, the various possible supernumerary bones. Every radiogram is beautifully reproduced and accompanied by an outline diagram in which all the special points are emphasized.

The second part, dealing with abnormal conditions, is in two volumes, the first being concerned with fractures and other injuries. Those regions specially prone to injury are most profusely illustrated; thus there are no fewer than twenty-nine radiograms of the fractures in or near the wrist, and every one of these is accompanied by a very clear outline diagram. For the same reason all the injuries near the large joints are generously illustrated. Fractures and dislocations of the spine are well figured. The figures of the fractures of the skull might well be increased in number and variety. There is a short section on gunshot injuries of bones and a few illustrations showing the different density of callus at periods varying from ten to eighty days. We hope the next edition will contain more figures showing varieties of delayed union, non-union, and pseudarthrosis.

The second volume of the second part illustrates the various diseases of the bones and joints. We confess that we have been a little disappointed in this because it does not approach the first two parts (normal skeleton and traumatic lesions) in completeness. Perhaps this is inevitable because the range of the subject is so much greater in the matter of diseased conditions. The section on tuberculosis of the bones and joints, forming nearly half the volume, is good and the figures are well chosen and typical. Syphilis is only scantily illustrated. New growths are shown in seventeen figures, which are quite inadequate for the subject. The rest of the book treats of various conditions, osteomyelitis, arthritis, dystrophies, and congenital malformations, and is full of good pictures of these conditions.

The conception of this book is very good, and it will form an invaluable work of reference. We foresee that the authors will soon have an opportunity of enlarging the section dealing with diseased conditions on the lines we have indicated.

Röntgenuntersuchung und Strahlenbehandlung der Speiseröhre. By Dr. JOSEF PALUGYAY (Vienna). Handbuch der Röntgenkunde, Vol. III. Royal 8vo. Pp. 392 + xiv, with 224 illustrations. 1931. Vienna: Julius Springer. Paper covers, RM. 56; bound, RM. 59.60.

THIS book bears the hall-mark of the publishing house of Julius Springer of Vienna. The author's main object has been to study by radiology the œsophagus in health and disease. Experiments have been performed by radiological means with the subject in the horizontal position and with the pelvis raised; the knowledge obtained has been applied to the physiology and pathology of the œsophagus. One chapter is devoted to the technique of this research. The author has not confined himself to radiology, but has presented a consideration of the pathology, symptoms and signs, differential diagnosis, and treatment of the various abnormalities of the œsophagus. The book is arranged in an orderly manner, and opens with an historical section. Some of the most important facts are re-stated in connection with the anatomy and physiology of the œsophagus. Attention is drawn to the variations in length of the œsophagus in different individuals and in the two sexes: greater variations are stated to occur in width. Interesting stereographic diagrams are given of transverse sections of the œsophagus in different regions with inspiration and expiration. In view of the present-day prominence of the sympathetic nervous system, the section on the nervous connections of the œsophagus is disappointing. The physiology of deglutition is considered fully and all theories are examined; the author reaches the conclusion that the act is very complicated, involving the co-ordination of the musculature of the pharynx, œsophagus, cardia, and diaphragm, and he disagrees with Magendie's division into three parts. Careful study has been made of deglutition with the body horizontal and also with the pelvis raised, and interesting conclusions have been reached.

Functional diseases of the œsophagus, with special regard to the radiological appearances, receive due attention, and no detail, however small, escapes the author's

attention, which is merited when we consider our ignorance of these subjects. The subject of cardiospasm is dealt with comprehensively with due regard to a consideration of the pathology. No statement, however, is made on the possible involvement of the sympathetic nervous system in this condition. A very useful description of the many varied radiological appearances in this condition is given, and the author states that it is possible to estimate the thickness of the cardia by radiography.

Organic diseases occupy a large portion of the book. An interesting account of diverticula is given—pulsion diverticula being divided into pharyngo-œsophageal (Zenker's) and œsophageal pulsion diverticula, which may occur anywhere in the œsophagus, according to the author. He states that there are two varieties of traction diverticula—first, there is the type produced by traction outside the wall plus pressure from within the lumen; secondly, the rudimentary type caused by adhesions of the œsophageal wall to its environment, hindering contraction of the wall.

Many clear radiological pictures of carcinoma of the œsophagus are given, and we are glad to see the statement made that carcinoma occurs in all parts of the œsophagus, which agrees with our experience. A section is devoted to œsophageal operations, together with a full consideration of the possibilities of the treatment by radium of carcinoma. The author states that carcinoma of the œsophagus in any other position than the cervical can only be treated by radium or X rays. The indications, contra-indications, and complications are discussed.

The whole work merits high praise. There is a good bibliography of the literature, the diagrams are clear, and the radiological pictures excellent.

Röntgendiagnostik der Gallenblase. By Priv.-Doz. Dr. F. Eisler and Dr. G. Kopstein. Radiologische praktika, Bd. XVII. Super royal 8vo. Pp. 153 + viii, with 151 illustrations. 1931. Leipzig: Georg Thieme. Bound, RM. 18.60.

This book keeps up the high standard already achieved in this series of works on radiology. The authors give a description of the anatomy and physiology of the biliary tract, pointing out how cholecystography has been of value in this sphere as well as in diagnosis. The technique of the oral and intravenous methods of cholecystography are described in detail. The radiological technique occupies very little space, and rightly so, the book being written from the point of view of interpretation of the radiograms, and correlation between the X-ray and clinical or operative findings. The reproductions of the numerous and excellent radiograms are good, the conditions referred to showing up well. There is a useful bibliography of over 350 references, which is an adequate summary of the growing literature on the subject.

Annals of Roentgenology: a Series of Monographic Atlases. Edited by James T. Case, M.D., Professor of Roentgenology, North-western University Medical College, Chicago. Volume VII, Urological Roentgenology. By Hugh H. Young, M.D., Clinical Professor of Urology, Johns Hopkins University; and Charles A. Waters, M.D., Associate in Clinical Roentgenology, Johns Hopkins University. Second edition revised. Large 4to. Pp. 564 + xlix, with 592 illustrations. 1931. New York: Paul B. Hoeber Inc. $20.00.

This is a very large book of some 564 pages; it is most handsomely printed and bound, and contains no fewer than 592 illustrations. In it will be found an enormous amount of information on almost every aspect of the surgery of the genito-urinary tract; it is by no means a mere atlas of skiagrams, for the text includes articles on the pathology, the diagnosis, and the treatment of the varied lesions of this tract. The authors' plan has been to discuss such subjects as tuberculosis, stone, and neoplasms of the genito-urinary tract and to illustrate them by skiagrams and drawings; accompanying these the case histories are invariably given.

Perhaps the most interesting of the chapters is that on arteriography of the aorta and its abdominal branches; for the purpose of showing the circulation of the kidneys 20 c.c. of 100 per cent solution of sodium iodide are injected into the aorta in the region of the first lumbar vertebra; apparently this can be done without

much risk, for the middle coat of the aorta produces prompt occlusion of the puncture. The resulting skiagrams are very striking, and show, for instance, the few vessels present in a tuberculous kidney as compared with those of the opposite organ. The authors sum up their present attitude to this novel method of investigation: "It is needless to say that one hesitates to express an opinion at this time upon this startling work by dos Santos and his confrères. Coming as it does from a man of marked scientific attainments, and with the assurance that puncture of the aorta has been done in 150 cases without untoward results, one must maintain an open mind, express the greatest interest in this remarkable presentation, and hope that future experience will bear out the conclusions of the authors."

We note that the authors have tried pyeloscopy and have abandoned it in favour of serial pyelograms; they consider that this method of investigation has little to add in the way of information and that it has some very distinct disadvantages. There is an excellent chapter on neurological lesions involving the urinary tract.

The book has one drawback, its rather high price, but we can recommend it as a real contribution to the surgery of the genito-urinary tract; it will, we think, be used chiefly as a book of reference.

On page 132, two skiagrams have been inserted upside down, but apart from this there are singularly few mistakes or misprints.

A Descriptive Atlas of Radiographs: An Aid to Modern Clinical Methods. By A. P. Bertwistle, M.B., Ch.B., F.R.C.S.E. Second edition, revised and enlarged. Crown 4to. Pp. 552 + xxviii, with 767 illustrations. 1932. London: Henry Kimpton. 42s. net.

The author is to be congratulated on his energy and diligence in bringing together this large collection of radiographs and on the fact that a second edition of the book has already been called for. The originals of most of the pictures have been supplied by various workers in special departments—Sir William Milligan, the nasal sinuses; Mr. Marxer, the dental; Professor Wilkie and Dr. Spriggs, the alimentary; Professor Fullerton and Mr. Kidd, the urinary; Professor Sicard and Mr. Dott, the nervous; and Mr. Morriston Davies, the chest.

The book constitutes a veritable storehouse of information and illustration. Our chief criticism is that the reproduction of the more complicated radiograms—those of the chest and abdomen, for example—is not clear enough to make these pictures of much value for the purpose of reference.

The Rational Treatment of Varicose Veins and Varicocele. By W. Turner Warwick, M.A., M.B. (Cantab.), F.R.C.S., Surgeon to Outpatients and Surgeon in charge of the Rectal Department, the Middlesex Hospital, London. Crown 8vo. Pp. 188, with 12 illustrations. 1931. London: Faber & Faber. 5s. net.

In addition to the treatment of varicose veins this book also gives a very exhaustive account of the anatomy and physiology of the veins of the lower extremity. The chapter on the etiology of varicose veins, of no fewer than 43 pages, deals most clearly and in great detail with the subject from the historical, experimental, and practical sides and is quite the best account of the subject we have seen. Although he describes the operations for varicose veins, the author considers them replaced by the injection treatment, which is described in detail.

The pathology and etiology of varicocele, as well as its treatment both by operation and injection, are described.

The author also gives an account of the injection treatment of piles. For first and second degree piles injection is the method of choice, and in those of the third degree it is worthy of a trial if, for any reason, operation is contra-indicated. The use of a 20 per cent solution of phenol in equal parts of glycerin and water is the solution preferred, and the injection of the solution into the base of the pile is emphasized.

To all who want a clear and simple account of the treatment of the three common conditions mentioned above, as well as those requiring a more academic account of varicose veins, this book should give all that is required.

Surgery. By S. K. Sen, L.R.C.S., L.R.C.P. (Edin.), L.R.F.P.S. (Glas.), late State Surgeon, Nepal. Vol. III. Surgery and Pathology of Growths. Demy 8vo. Pp. 348 + viii, with 56 plates. 1931. Calcutta and Burdwan: The Surgical Education Society Ltd., Rs. 7/8 per vol.

"This is the third of the series of eight volumes of Surgery, the whole plan of which is described in the preface of the first volume. It contains the description of all pathological Regenerations and New Formations. The book is intended for those mid-senior students who have just finished a course of Pathology, and are attending Surgery classes.

"It is written to the standard of qualifying examinations in England. Being a student of Edinburgh I have mainly followed the teachings of the Edinburgh School of Medicine where I attend the ordinary classes even now when opportunity offers itself. In fact many pages are written from the lecture notes taken by me in the University classes very recently."

These first two paragraphs of the Preface explain clearly the intention of the author. The book is fairly well illustrated with plates made from the author's own cases. It is quite likely that a book written in this way will be more useful to the Indian student than the average European text-book.

Minor Surgery. By Lionel R. Fifield, Late Surgical First Assistant and Registrar, London Hospital. Second edition, revised by R. J. McNeill Love, M.S. (Lond.), F.R.C.S., Surgeon, the Royal Northern Hospital, etc. Crown 8vo. Pp. 440 + viii, with 281 illustrations. 1931. London. H. K. Lewis & Co. Ltd. 12s. 6d. net.

The second edition of this well-known handbook contains few alterations or additions. It is a thoroughly sound book to give to a student beginning surgery, containing, as it does, such often neglected topics as how to pass a catheter, apply a plaster, or extract a tooth. The use of tannic acid for burns, the injection treatment of varicose veins, and the methods of treating minor affections of the ear, are welcome additions. The author will not receive a general acceptance of his theory that Volkmann's contracture is "caused by an extravasation of blood within the sheaths of the flexor muscles". If any serious criticism must be made, it is that many methods of treatment are given without sufficient stress upon the principles which should govern their selection.

Surgical Pathology of Prostatic Obstructions. By Alexander Randall, M.A., M.D., Professor of Urology, University of Pennyslvania. Large 8vo. Pp. 266 + xiii, with 78 plates. 1931. London: Baillière, Tindall & Cox. 37s. 6d. net.

This monograph is an important contribution to the literature of urology; it is the result of ten years' study of the autopsies at the Philadelphia General Hospital, an institution with over two thousand beds and a mortuary in which two or more autopsies are performed daily. During this period 1218 specimens of the male bladder and prostate were examined, and later their case histories were obtained and correlated. Hardly any of these cases of prostatic obstruction had been operated on, and this is explained by the fact that the Philadelphia General Hospital, in addition to the usual medical and surgical divisions, comprises departments for tuberculous and insane patients and "also functions as the city almshouse and home for the indigent".

The author describes and illustrates the gross morbid pathology of the various affections of the prostate—hypertrophy, median bar, carcinoma, and abscess—and he discusses the surgical handling of the obstruction produced by such lesions. There are numerous plates showing the various morbid conditions of the prostate; they are all photographic, and, whilst perhaps they are as good as modern photography can produce, some of them fail to satisfy the reader, and they leave a feeling that Professor Randall would have been better advised to have given fewer plates and to have had them drawn by a competent artist.

One point of interest is that the negro population forms no less than 30 per cent

of the admissions to this great hospital; they not only suffer from prostatic obstruction but they suffer from it at an earlier age than the whites; this confirms what Freyer affirmed as the result of his experience in India, that the black races are really ten years older than their age as compared with the white.

As the outcome of his studies of benign hypertrophy, the author divides prostatic enlargement into no fewer than eight varieties; we consider this subdivision unnecessary, for almost certainly they are merely stages in the development of this disease. From an examination of seventeen specimens of carcinoma of the prostate he concludes that there is no foundation for the statement made by Tandler and Zuckerkandl that this disease starts usually in the posterior lobe; and again, though thirteen out of the seventeen had metastases, there were only four instances of secondary deposits in bone.

Altogether this book is well worth reading by those who are interested in the surgery of the prostate.

Surgical Pathology of the Diseases of Bones. By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 272 + xiv, with 211 illustrations. 1930. London: J. B. Lippincott Co. 21s. net.

THIS is one of a series of monographs by Dr. Hertzler on special surgical pathological subjects. He gives a clear description of all bone diseases, with a good bibliography of the classical articles on each subject at the end of every chapter. On the many points of controversy in bone pathology the author does not, in most cases, express his opinion but gives a review of those held by other authorities. For tumours he wisely adopts the classification of the American registry of bone tumours. The chapter on diseases of unknown origin, dealing with such conditions as Perthes', Köhler's, Schlatter's diseases, etc., and fragilitas ossium, gives us no new help; and indeed no reference is made to calcium metabolism or lesions of the parathyroid glands in the description of osteitis fibrosa cystica. The illustrations are mostly very good, but some of the skiagrams are not of very typical specimens; for example, there is not shown a really typical skiagram of a benign giant-celled tumour.

This book should be useful for those who want to obtain a broad knowledge of the pathology of bone diseases, but for those requiring special knowledge on some particular condition there is hardly enough detail given.

The Laboratory in Surgical Practice. By C. E. DODDS, M.V.O., M.D., Courtauld Professor of Biochemistry in the University of London, etc.; and LIONEL E. H. WHITBY, C.V.O., M.D. (Camb.), M.R.C.P. (Lond.), D.P.H., Bacteriologist to the Middlesex Hospital, Bland-Sutton Institute of Pathology, etc. Demy 8vo. Pp. 187 + x. Illustrated. 1931. London: Constable & Co. 8s. 6d. net.

THIS is the first of a new series of surgical monographs under the general editorship of Gordon Taylor. It describes in clear and simple fashion the numerous laboratory tests which are employed in surgical work, and includes such clinical methods as intravenous pyelography. It will be useful to the actual laboratory worker, but should be read by surgeons who call for these tests. The large experience of the authors in such investigations is an assurance of the reliability of their statements as to what can and what cannot be expected from them. The book is well printed, easy to read, and has a number of useful diagrams.

Schmerzverhütung: Zwölf Vorlesungen. By Dr. FRITZ STARLINGER (Vienna). Medium 8vo. Pp. 106 + vi. 1931. Vienna: Julius Springer. RM. 6.60.

THIS is a small book, divided into twelve chapters, on various methods of the prevention of pain, by the Assistant in the Eiselsberg Clinic of the University of Vienna. It is written in German for the use of students and young doctors, and contains articles on various forms of ether narcotics and forms of anæsthesia.

Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. Vol. LXXXI (Vol. XI, Fourth Series). No. 3. July, 1931. Royal 8vo. Pp. 253–378. Illustrated. 1931. London: The Lancet Ltd. Single numbers, 12s. 6d. net; annual subscription, £2 2s. net.

THIS volume contains a description of a study of osteitis fibrosa cystica associated with tumour of the parathyroid, and a detailed report of a case is given. The size of the tumour was 2·7 by 2·2 by 2 cm., and the outstanding feature of the case was hypercalcæmia. In the same volume there is another short article on the condition of the teeth of this patient. It appears that the growth of the teeth ceased soon after the age of 15 years, or became much retarded, the result being analogous to the arrested growth of the long bones. There is a remarkable contrast between the rarefaction of the bones and the comparative integrity of the teeth.

Specific Changes in the Blood Serum. A Contribution to the Serological Diagnosis of Cancer and Tuberculosis. By S. G. T. BENDIEN, Serological Laboratory, Zeist, Holland. Translated by A. PINEY, M.D., Director of the Pathological Department, the Cancer Hospital, London. Royal 8vo. Pp. 95 + xii, with 64 illustrations and 5 spectra in the text and on 8 plates. 1931. London: Wm. Heinemann (Medical Books) Ltd. 10s. 6d. net.

THIS is a very excellent translation of a book which contains a great deal of highly technical material and is therefore of greater interest to the serologist than to the clinician, who, at the present stage, must be largely guided by the findings of other laboratory workers in their attempts accurately to evaluate the author's various claims and theories in connection with cancer. It cannot, in fact, be said that this particular book makes these theories entirely clear, but as Dr. Bendien explains in his preface that he is only at the beginning of the way and that his conclusions are probably not absolutely correct, his book must be read and judged with these facts in view. In any case he gives the necessary details with the aid of which others may carry out the flocculation test, which, it is claimed, may give guidance in cases of tuberculosis and carcinoma. How much practical assistance to clinicians will be forthcoming must be decided by the extent to which other investigators are able to confirm the author's experimental results, particularly in their relation to clinical conditions.

L'Ostéosynthèse des Os longs. Etude critique, biologique et pratique. By C. E. CORNIOLEY (Geneva). Preface by Prof. CH. JULLIARD. Royal 8vo. Pp. 422 + xvi, with 102 illustrations in the text and 16 plates. 1931. Paris: G. Doin et Cie. Fr. 75.

THIS monograph represents a very fair and able description of the author's experience in the operative treatment of fractures, gained in twenty-six years' experience. A very great number of different methods of fixation of the bones are described, and some of these are illustrated by means of line diagrams; but neither the descriptions nor the illustrations would suffice to enable the method to be carried out by these instructions. The reaction of the living bone towards metallic suture material is well illustrated, but the fundamental necessity of mechanical efficiency in fixation is not made equally clear. Throughout the whole book, and especially in the chapter on forearm fractures, there are many cases quoted and illustrated in which the metallic fixation is obviously inadequate to secure immobilization. The osteo-periosteal graft is mentioned, and the cases figured in which it has been used serve only to prove its inefficiency. It is sad to see the operative treatment of fractured neck of the femur only illustrated by the long carpenter's screw method.

The book does not profess to describe other methods of fracture treatment besides those of open operation, but a certain number of splints are mentioned and their uses discussed. The very great importance of skeletal traction and its use as an indispensable preliminary to open operation is not given the place it deserves.

BOOK NOTICES.

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

United States Army X-ray Manual. Authorized by the Surgeon-General of the Army. Second edition, rewritten and edited by Lieut.-Colonel H. C. PILLSBURY, M.D., U.S.A. Crown 8vo. Pp. 482 + xvii, with 228 illustrations. 1932. London: H. K. Lewis & Co. Ltd. 25s. net.

Ikonographia urologica. Edited by ALFRED ROTHSCHILD (Berlin); VICTOR BLUM (Vienna); FRIEDRICH NECKER (Vienna). Part IV. Pp. 21, with 25 illustrations. 1932. Berlin: Georg Stilke. RM. 5.40.

The Technique of the Non-padded Plaster Cast. By FRITZ SCHNEK, M.D. (Vienna). With a Preface by LORENZ BÖHLER, M.D. (Vienna). Translated by DOUGLAS D. TOFFELMIER, M.D. (Oaklands, California). Royal 8vo. Pp. 140 + viii, with 169 illustrations. 1932. Vienna: Wilhelm Maudrich. $5 post free.

Technique de l'Ostéosynthèse. By ROBERT DANIS, Professeur de Clinique chirurgicale à l'Université de Bruxelles. Royal 8vo. Pp. 162, with 149 illustrations. 1932. Paris: Masson et Cie. Fr. 55.

Konstitution und Vererbung in der Orthopädie. By Prof. Dr. B. VALENTIN (Hannover-Kleefeld). Royal 8vo. Pp. 30, with 20 illustrations. 1932. Stuttgart: Ferdinand Enke. RM. 2.50.

Der heutige Stand der Knochenbruchbehandlung. By MORITZ BORCHARDT. Hefte zur Unfallheilkunde, Heft 11. Royal 8vo. Pp. 72, with 30 illustrations. 1932. Berlin: F. C. W. Vogel. RM. 7.80.

Handbuch der Unfallmedizin. By Dr. CONSTANTIN KAUFMANN (Zürich). Vol. I. Allgemeiner Teil. Unfallverletzungen. Royal 8vo. Pp. 862 + xxviii. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 52; bound, RM. 55.

Tuberculose osseuse et ostéo-articulaire. By ETIENNE SORREL, Chirurgien de l'Hôpital Trousseau, and Mme. SORREL-DEJERINE, Ancien Interne des Hôpitaux de Paris. Royal 4to. Pp. 514, with 640 illustrations. 1932. Paris: Masson et Cie. Fr. 350.

Surgical Pathology of the Female Genital Organs. By ARTHUR E. HERTZLER, M.D., Professor of Surgery, University of Kansas. Hertzler's Monographs on Surgical Pathology. Royal 8vo. Pp. 346 + xxii, with 285 illustrations. 1932. London: J. B. Lippincott Co. 21s. net.

Tratado de Patología quirúrgica general. By Dr. MANUEL BASTOS ANSART (Madrid). Large 8vo. Pp. 853 + xx, with 473 illustrations. 1932. Barcelona: Editorial Labor, S.A. No price given.

Spezielle Chirurgie der Gehirnkrankheiten. Edited by Prof. FEDOR KRAUSE. Vol. II/2, Die epileptischen Erkrankungen, by Prof. FEDOR KRAUSE and Dr. HEINRICH SCHUM. Royal 8vo. Pp. 523–957 + xx, with 95 illustrations. 1932. Stuttgart: Ferdinand Enke. Paper covers, RM. 63; bound, RM. 65.50.

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